Vol. I APRIL, 1917 No. 2

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THE AMERICAN REVIEW OF TUBERCULOSIS

JOURNAL OF

THE NATIONAL ASSOCIATION FOR THE STUDY AND PREVENTION OF TUBERCULOSIS

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PUBLISHED MONTHLY

AT 2419-21 GREENMOUNT AVE., BALTIMORE, MD.

BY THE NATIONAL ASSOCIATION FOR THE STUDY AND PREVENTION OF TUBERCULOSIS

Entered as second class matter March 9, 1917, at the post office at Baltimore, Maryland, under the Act of August 24, 1912

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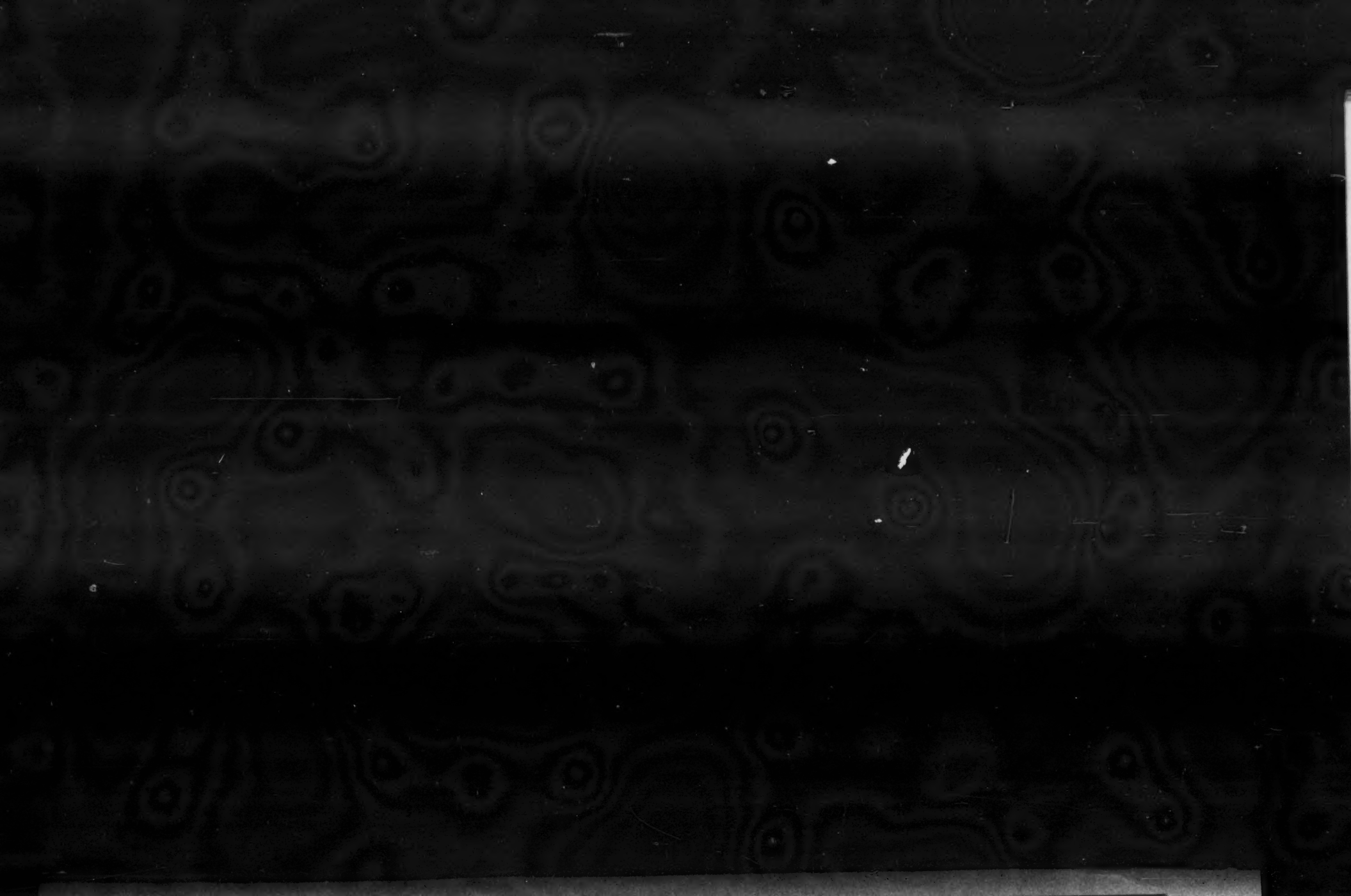

THE NATURE OF RESISTANCE TO TUBERCULOSIS[1]

ALLEN K. KRAUSE

From the Kenneth Dows Fund for the Study of Tuberculosis, of the Medical Clinic of the Johns Hopkins Hospital, Baltimore, Maryland

As our insight penetrates deeper and deeper into recesses that were once dark to us we consciously or unconsciously demand more precise description of terms to denote phenomena that obtrude upon our senses but defy our understanding. Upon first appreciating a thing, be it light or sound, an abnormal sensation or an unusual conformation of the body, we give it a name. But we are mentally so endowed that we are not long content with the mere name of a thing. We must know where and how it begins and ends, and through what media it works; we must discover its attributes and, these determined, we must enlarge and refine our definition and description. With more precise definition and description comes the conception that nothing that we sense is isolated or autogenic. It is born of something and brings forth something. And once our minds begin to deal with its causes and effects then we can say that the thing has entered into our understanding. Then only can we affirm that its name is to us perhaps something more than a mouthful of words.

Terminology, description and a knowledge of cause and effect: every domain of knowledge, every phase of science passes through these three stages on the road from sensation to reason; and medicine, the all-embracing, the focus of all sciences, the handmaid of mathematics and physics and chemistry and botany and climatology and sociology and psychology, is today perhaps in all three stages. A genius, a Helmholtz, arises and lays bare the mechanism of the eye. A Richard Bright, a Louis or a Gerhard sifts and describes and clarifies a confused mélange. A man with a knack for observation, perhaps a dermatologist, classifies those things that are red, or those that are raised, or those that itch or those that appear on certain parts of the body; and he thereby really brings under control his science and takes a step far

[1] Read before the Academy of Medicine, New York City, February 15, 1917.

beyond the time when he called everything a "humor" or a "tumor." But terms, mere terms, persist, and are too often accepted as explanations. Nephritis, cardio-nephritic, myocarditis, arterio-sclerosis, hypertension, hypertensive-nephritic, myocardial insufficiency with hypertension, hypertension with renal insufficiency; what a real evolution of knowledge these terms convey, yet how little more than terms they are! Naming much and describing some things they explain nothing.

I feel very diffident in coming before you to-night and talking about what has often seemed to me to be, as commonly used, a mere term or symbol. I have attended clinics given by some of our best men and have heard the affirmation that a liberal diet of milk and eggs would increase the tuberculous patient's resistance. I have seen the specialist impressively produce his syringe and just as impressively assure the patient that a prolonged course of tuberculin therapy would further the latter's resistance. Time and again I have read that a man overcame his tuberculosis because his resistance improved or that he declined because he had no resistance. And hearing these statements I have often wondered just what was going through the man's mind when he talked about resistance. Did he mean anything; or, if he did, had he any conception of what he tried to express? Or did he merely wish to avoid mental exertion and therefore seek to explain an effect by a handy word?

If there is such a thing as resistance to tuberculosis then we must mean that the human body can protect itself in such a way that the tubercle bacillus, a parasitic invader, can gain no foothold or make but limited headway in it. We must assume further than under the influence of a specific irritant, the bacillus, the body reacts in a particular manner. So long as it reacts sufficiently to prevent invasion or spread of the tubercle bacillus just so long we would say that resistance is satisfactory. If however it fails to do so then resistance is lowered or absent. The entire relation of the tubercle bacillus with the animal organism is one of reaction. Vivre c'est réagir: to live is to react; and from conception to final dissolution man's vital history is nothing but an unceasing period of countless myriads of reactions that go on simultaneously or unfold themselves in sequence. We must look upon every reaction to a potentially harmful stimulus or irritation as an effort at protection and adaptation. The body reacts to the rays of the sun in a certain definite manner and thus protects itself against them. It reacts to the introduction of pork or morphine in wholly different

ways and thus adapts itself to them. It reacts to the implantation of the tubercle bacillus in a very particular manner and meets the germ with the formation of a special type of cellular accumulation which we call the tubercle. These are trite though fundamental conceptions of biology. In thinking of any tissue or organic attributes like resistance we dare not forget them, and it is likely that during the next few minutes I shall recur to them again and again. To live is to react. And, if the stimulus or irritant be one that is disagreeable to tissue then to live is to resist. Whether resistance to a given irritant at any given time is adequate enough is another matter, yet it is certainly true that if we are to live then we must resist. Therefore, in addition to the three necessary attributes of assimilation, motion and reproduction as they are generally given for protoplasm, I would add a fourth attribute, resistance. I am aware that this fourth attribute may be partly contingent on others such as assimilation and motion, but it is conceivable that in highly organized animals resistance goes on independently of them.

We assume therefore that there is a resistance to the tubercle bacillus but in thus stating the proposition we have got little beyond giving a name to the known fact that tissues react to tubercle bacilli. We have not explained or illuminated a single thing. It is my purpose to-night to inquire into the nature of this resistance, to attempt to discover how it acts and what are its effects. I trust that you will be tolerant with me while I lead you into regions which, I know only too well, are strewn with obstacles and beset with pitfalls.

Let us first look into what happens when tubercle bacilli are taken into the body for the first time. Let us take for granted what happens in the vast majority of cases, that is, that they enter by the mouth or the nose. After excursions of varying length they come to rest. They may be caught in the vibrissae of the nose, on the moist surfaces of the tortuous nasal passages or in the mucus of the naso-pharynx, and be most immediately blown out or expectorated with the naso-pharyngeal secretion. Here at the very beginning we meet with a protective mechanism at the body's command.

But let us assume that they get further and penetrate the mucous membrane of the tonsil or pass into the submaxillary glands or go further down into the larynx and reach the deep cervical glands. They are now no longer ON an epithelial surface where they cannot produce an effect: they are IN tissue. What happens now? The tissue reacts.

Fixed tissue cells of connective tissue (mesoblastic) type begin to show agitation and unrest. Their nuclei swell, chromatin rearranges itself and heaps up in a bizarre manner, the cells themselves enlarge and in a short time we find that they are dividing, multiplying and heaping up, and surrounding and englobing the offending parasites. Now, too, cells begin to slip in from the circulation. Leucocytes accumulate in this new, round, globoid speck of matter and a tubercle comes to view. A few more days go by, and we meanwhile study the life cycle of this tubercle from time to time. What do we find? We notice developing under our eyes an avascular growth or formation of tissue, surrounding and englobing the parasitic invaders and shutting them off from the rest of the body. The bacilli are held within a globular bloodless wall. So long as the wall is competent, so long as circulation between the interior and exterior of the tubercle is at a minimum, so long as the fibrous wall of the tubercle holds against invasion by bacilli from within or a flooding of body fluids and a consequent softening from without, just so long will the body have the germs in this initial tubercle under control.

Fortunately for most of us this is the extent of our more intimate and involuntary acquaintance with the tubercle bacilli. We are infected but without clinical disease. We have tubercle but not tuberculosis. And once having tubercle, it may be that with never a symptom of specific illness we will go through a long life without ever thoroughly eradicating the tubercle bacilli from our bodies. Though this statement cannot be proved, all our knowledge points to the fact that once having gained entrance to an animal body and once having developed sufficiently to arouse tubercle, the bacilli are never completely wiped out. Why then are all of us infected while so few of us are ill? Where does resistance begin, what does it consist in and how far does it go? For surely there must be some resistance or otherwise the bacilli would multiply and spread through us like wildfire. Under the very special premises that I have outlined, is there a resistance to infection or implantation or is the resistance merely to subsequent invasion? Has any man or animal a native resistance to initial infection by a type of tubercle bacillus to which his kind is susceptible?

We know that several diseases do not decimate civilized communities in the way in which they cut down primitive peoples. Measles and tuberculosis are real plagues if introduced among the Esquimaux and the dwellers of the South Sea Islands, but today occur as relatively

benign diseases among peoples who have a long history of community life behind them. For this reason we postulate and teach that some nations have developed a racial immunity to tuberculosis and measles. This may be true to a certain extent but this racial resistance is not a resistance to infection. If exposed, practically every child on earth today develops measles and practically every human being reacts with tubercle. In other words, all become infected; and we cannot assume that there is any native resistance to implantation of the microörganism. Again, if an individual is not tainted with tubercle we cannot by any known method demonstrate that he harbors substances that may be antagonistic to the tubercle bacillus. And if we treat an animal by certain procedures we may find in his blood certain bodies like agglutinins, aggressins, precipitins and opsonins, yet unless we give the animal anatomic tubercle it will not be more resistant to infection than any other non-infected animal that has not been thus treated. Because of these facts we must assume that man and animals have no native resistance to infection by tubercle bacilli in the sense that born in them is some substance or substances that specifically destroy or neutralize tubercle bacilli that seek an entrance. But there is a difference between the way in which the tissues of the Jew meet the onslaught of the tubercle bacillus and that in which the tissues of the Esquimaux meet it. There is a difference between the reaction of the tissues of the newborn infant and those of the city dwelling adult to bacillary implantation. Both the time and character of these reactions are different. The tubercles develop differently and this difference of development in large part determines the issue. Remember that tubercle formation is response to irritation, be the irritant a hair, a grain of sand or a living, self-propagating, parasitic tubercle bacillus containing as it does a very high percentage of fats and waxes that are got rid of by the body only with great difficulty. This reaction is resistance, and an attempt on the part of the body to isolate or wall off the foreign body. Remember that inasmuch as all of us become harborers of tubercle we must assume that there is no native *increased* resistance to infection by tubercle bacilli. Remember too, that as tissues become infected their capacity to react changes.

Let us now consider for a moment what I choose to call the mechanical element in resistance to tuberculosis. I have often wondered why pathologists have not paid more attention to the architecture of the tubercle as a formation designed by its very structure to resist further

invasion by the tubercle bacillus. To me the most impressive feature of a tubercle is the fact that here we have a wall built around a foreign body, that the wall is formed around the foreign body to encyst it and render it innocuous to the infected organism, that as long as the wall holds the germs cannot get out and spread throughout the organism, and that a necessary condition to the competence of the investing wall is fibrosis and a minimum of circulatory give and take between the centre of the tubercle and the tissue that surrounds it. We can go even further. We can affirm that to produce symptoms of disease the tubercle must give up something in itself to the body, that the latter must absorb this material if it is to suffer symptoms of illness, that it can absorb something from the tubercle only if there is again a sufficient circulatory give and take between it and the tubercle, and that the circulatory exchange between tubercle and infected organism will be directly proportionate to the degree of fibrosis or sclerosis of the investing wall. Thus resistance to further spread of bacilli and freedom from symptoms are to be understood not in terms of the body producing some fighting units which go out to battle with the germs or to neutralize their poisons. They are to be understood as being simply the result of a mechanical barrier which the body interposes between itself and the parasite.

Suppose a man has such a tubercle in his lung in close proximity to a blood vessel. It has never made itself manifest. The man has always been in perfect health. In perfect health he runs a hard race or plays a fast tennis game. Like a bolt from the sky a pulmonary haemorrhage strikes him down, and immediately he falls ill. Another man leaves home in the morning feeling perfectly well. Suppose that for years he has had a slow and sluggishly progressive tuberculous process in a bronchial gland, yet has never had a symptom from it. Suppose further that while he is at his desk the last tenuous sheath between this caseous gland and the lumen of his bronchus is broken through, perhaps because of coughing or sudden muscular effort. And suppose that the contents of this diseased gland are discharged widespread into his bronchus. Such a man comes home in the evening sick, with beginning caseous pneumonia.

Suppose now that for years a small gland lay close to the thoracic duct and that firmly shut within its capsule there smouldered tubercle. But the time comes when the tuberculosis ulcerates into the thoracic

duct and from perfect health a man is precipitated into generalized miliary tuberculosis.

What shall we say about such patients? Shall we say that they developed acute tuberculosis because their resistance gave way? Yes, we may say so. But what did their resistance consist of? In these cases resistance certainly meant that foci of infection and disease were isolated and shut in by barriers of tissue, and that when the barrier went, resistance went.

Let us take another instance. Here is a tubercle in the lung, with bacilli well shut in and wall thoroughly competent under ordinary conditions, and its possessor a thoroughly healthy man. He develops a common cold or a bronchitis and soon after presents symptoms of tuberculosis. In terms of resistance how shall we view the onset of his tuberculosis? I have often heard just such cases explained in terms something like this: that the acute respiratory disease lowered the man's resistance, that he was left in a run down condition, that the cells of his body therefore lay more open to invasion by the tubercle bacillus. I shall not pay detailed attention to the lack of precise reasoning in such an explanation. But I should like to put forth one that is much more plausible, and, I believe, deals more with physiological facts. Is it not more likely that during the man's "cold" or bronchitis there was a certain amount of congestion of the affected tissues, that this congestion extended to the neighborhood of the tubercle and that it established new channels of communication and circulation between the tubercle and the surrounding tissue, unlocking or opening the capsule and allowing a fresh dissemination of interned bacilli? If hard exercise will produce the circulatory conditions necessary to break through an old tubercle and thus cause unlooked for hemorrhage, why will not congestion about a tubercle exert a more or less similar effect? Here again resistance is a matter of simple mechanics. The patient is as resistant as the shell of his tubercle.

Hard physical exercise, sudden and intense muscular exertion, a cough, a common cold, childbirth, tuberculin in overdose, alcohol, all can produce the same circulatory effect in the neighborhood of the tubercle and, theoretically at least, can bring about the same result, a better circulatory give and take between the tubercle and the host with a recrudescence of symptoms or a mobilization of bacilli or both. This may be momentary or it may be prolonged. If the inflammatory

stimulation is not overdone, proliferation and repair will soon set in and good may result. If it is too intense, then dissemination and progression of disease may ensue. But there is no doubt that what we have been accustomed to call resistance to tuberculosis is more or less bound up with the mechanics of the circulation around the tubercle.

The body responds to exercise in a very definite way: the pulse rate and pressure rise and the respiratory activity is increased. It is not difficult to imagine how under these circumstances the lungs are affected and what might happen to old encysted tubercle. We recognize this fact in treatment, particularly when we warn our patients to suppress cough as much as possible. By complete rest we put the lungs in as complete circulatory inactivity, relatively speaking, as possible, and we do raise the patient's resistance; but we do this in a mechanical way by striving to avoid any manoeuvre that will enhance the circulation of the lung and of the tissue around the tubercle.

Consider too the accidents that must modify the course that tuberculous infection and disease are going to take. In our struggle to attain definite rationale and generalization in medicine I sometimes think that we are all too prone to forget the large part that accident plays in the development of a pathological process. A man may have vegetations on his heart valves and these may be continually shed off and lodge in skeletal muscles and no harm result. But another man with an identical cardiac condition may drop dead because the first embolus that broke loose was swept into his coronary artery. Similarly, accidents undoubtedly determine the course of tuberculosis. It surely makes a great deal of difference whether the first infection in the lungs develops near the pleura or near the hilus or near a large blood vessel or at the apex. The brain is not the only organ of the body that has its silent areas and its eloquent areas. The lungs have theirs too: and the effect will be not so much a matter of resistance as of location. To say that a man who had a caseous gland that erupted into the thoracic duct got generalized miliary tuberculosis because he lacked resistance to tuberculosis would be just as foolish as to affirm that a man who dropped dead lacked resistance to coronary embolism.

Besides the accidents of place or location there are the accidents of time. A great deal may depend on just when a patient develops an intercurrent infection such as an acute bronchitis, or suffers unusual physical strain. The same episode in his life may produce very different results. It may occur when the concealed tubercle is thoroughly

sclerotic and therefore completely withstands the changed anatomical or physiological processes going on in its neighborhood. Or it may occur at a time when the investing wall of the tubercle has just the proper structure to establish fresh communications with the surrounding tissue under the right conditions. Perhaps this is why young children if compared with adults withstand stress and infections relatively poorly so far as the effect of these on quiescent tubercle is concerned. Their tubercle is of comparatively recent origin, is not completely fibrosed or healed and is thus much more open to assault. As they grow older this healing process has advanced further and further so that we say that in regard to tuberculosis five to fifteen years is the golden age of man's span. But once having passed puberty they enter a period of from fifteen to twenty years when life's stresses are at their maximum and the increased mental and physical strain in many cases produces local effects that are more than even healed tubercle will bear. I say "healed" tubercle with some mental reservation. I would have you recall that living tubercle bacilli have been found in the chalky deposits of tuberculous areas that had healed by calcification. It is for these reasons that I am very fond of telling our students that the development of tuberculous disease from old benign tubercle depends largely on whether the patient gets a cold or rows a race or becomes pregnant at the wrong time.

We have thus far discussed the protective reaction by which the tissues seek to hem in and wall off the first tubercle bacilli that gain entrance to the body and exert their effect upon them. We have also considered local conditions by which the infected organism resists the development of tuberculosis. We have looked upon the tubercle as a growth the periphery of which acts as a barrier to the egress of bacilli yet is subject to all the physiological disturbance that goes on around it. We have seen how the permanence of benign tubercle or the conversion of benign tubercle into tuberculosis will depend largely on the mechanical interaction and balance of these opposing factors. Let us now extend our line of reasoning to an inquiry into conditions in the patient who presents himself to us with active tuberculosis.

Such a patient, of course, has anatomic change and he has symptoms. Why has he symptoms? Because he is absorbing something from his foci of disease. Why does he absorb something? Because there is a sufficient circulatory give and take between his foci and the surrounding tissue. Why is there this circulatory give and take? Because the

walls, the envelopes, the investments of these foci are not competent to block off the interior of the tubercles. If, in course of time, these walls become more and more fibrous and more and more impervious the severity of the patient's symptoms will surely diminish. This fact is so obvious that I consider it hardly necessary to enlarge upon it. Whether by design or not we recognize this principle in treating our patients. If they are symptomatic we put them at rest in order to quiet the circulation and the respiratory activity. Under such conditions they may be without symptoms. If now we allow them to exercise again symptoms immediately recur. Now their relief from symptoms, their resistance to symptoms, cannot be due to any neutralizing substance which the body elaborates, for, if it is, then we should not expect this prompt response to rest and exercise which by their action on respiration and the circulation we know simply diminish or increase the opportunities for absorption. You have all had patients with incipient tuberculosis who suffered markedly from symptoms of absorption and who, upon the ulceration of their process, informed you that they felt a great deal better. Here by ulceration Nature has applied the prime surgical principle of more or less thorough evacuation of a focus of disease.

The point I want to make is that the presence or absence of symptoms is to a certain extent contingent on focal mechanics. I think we can say absolutely that increasing fibrosis means a gradual release from symptoms, while increasing softening without coincident and sufficient peripheral fibrosis means added severity of symptoms. The man who has extensive anatomic tubercle with comparative freedom from symptoms has his tuberculous areas well sclerosed and hemmed in. The man who has but slight anatomic change with marked constitutional disturbance has but very slight investment of his focus. Whether this mechanical element is all that there is to resistance to symptoms I am not prepared to say, but it certainly plays a very large part in resistance.

And, as far as extension of anatomic change is concerned, you can neglect all other factors and assert almost dogmatically that so long as fibrosis is the predominant element in any particular case, just so long are fresh eruptions much less likely to occur than if degeneration and exudation gained the upper hand. Apart from any native or acquired habit of bodily constitution which, in a vague way, we are pleased to call resistance, purely local mechanical conditions play a very large part in resistance to tuberculosis, whether against first infection or against subsequent eruption.

I am, of course, aware that you are all saying, "Yes, but what determines fibrosis and caseation, degeneration and repair? Is it not possible that there is an underlying resistance, a native or acquired attribute of the animal body, that confers upon the tissues the ability to react in such a manner that ideal fibrosis is attained?" Yes, it is not only possible but highly probable, and it is to this phase of resistance that I wish to address myself during the remainder of our inquiry. The manner in which tissues react determines resistance. Let us now examine the probable factors that determine or influence the reactions of tissues. Let us study the reaction of tissues to irritation in general and to the tubercle bacillus in particular.

I take it as axiomatic that individuals vary tremendously in their reaction to irritation. This ability to react in varying degrees is certainly to some extent congenital. Stimulated by the same irritant one man's cells will overreact while another man's will hardly respond at all. In this connection Maud Slye's work is of tremendous significance. She works with two large series of mice. The one series consists of animals that have never had cancer bred into them; the other, of those which she is continually fortifying with a cancer heredity by mating their progenitors with individuals of known cancerous stock. She finds that individuals of the cancer strains react very differently to irritation than do those of clean stocks. If injured, many will develop cancer at the site of the wound while none of the non-cancerous strain ever reacts with cancer under the same conditions. Many of the latter, however, become ill with the ordinary infections and die. In cancerous strains, infections are very unusual and, if they occur, are resisted well. She would therefore generalize about as follows: Cancer is the result of a congenital tendency to overreaction to irritation. Animals with this tendency do not easily acquire infections. If they do develop them they resist them well. Infections strike down the weak. Cancer attacks the strong.

If Maud Slye's work is sound what shall we say? Shall we conclude that it is possible that the same tissue tendency that is responsible for cancer also protects against infection. Shall we assume that the irritant, the parasitic microörganism, gains slight foothold or none at all because it is immediately met by an exaggerated effort on the part of the body cells to ward it off? I am here reminded of a remark which that wonderfully keen observer, Dr. Trudeau, repeatedly made—that cancer is a disease of the strong, progressive tuberculosis a disease of the weak. He undoubtedly based this observation on his impres-

sion that so many people who developed cancer gave histories that were relatively free from infection during early life. I do not wish to develop this particular phase of the subject further. The point I would ask you to remember is that individuals do vary in their response to irritation, that this response may take the form of an overreaction, an overgrowth, an overproduction of tissue, that evidence is coming in that in some of us this tendency is inbred and that the same mechanism that resists infections may lay us open to cancer if the proper irritation is at work.

If any foreign body that is not assimilable gains access to the body the tissues react in a characteristic way, that is, with the formation of tubercle. Similarly, tubercle bacilli which are more or less non-assimilable by the tissues incite the formation of tubercle. Now there are several things that will modify the type of reaction that sets in against the irritant. One is the number of infecting bacilli. Long ago Baumgarten pointed out that if the bacilli were few the reaction would be manifested by the proliferation of only one type of cell. If only a few bacilli lodge at a point then for a number of days the only reaction which we note is a proliferation or renewed growth of those cells which we designate as belonging to the fixed tissue type. The resultant structure is a tubercle that has all the features of a tumor and none of the essentials of an inflammation. Such a tubercle may be made up of cells of the connective tissue type alone. There may not be a leucocyte or any other element of inflammation or exudation in it. Here we have really a growth or new growth or tumor, comparable to what under ordinary circumstances we would call a fibroma.

If, however, the initial number of tubercle bacilli is large, the first reaction is an outpouring of polymorphonuclear leucocytes. But within an hour the fixed tissue cells begin to show signs of unrest and proliferate. The polynuclear leucocytes soon disappear and tubercle formation goes on characteristically with a gradual inwandering of lymphocytic cells which distribute themselves throughout the mass of fixed tissue cells which in this location have come to be known as epithelioid. We see therefore that under ordinary conditions the body reacts to first infection in a variable manner according as the irritation is more or less intense. But the reaction is very regular and definite, and, if the infecting bacilli be the first that have gained access to the body, the result will always be the same. The reaction to first infection will always eventuate in the production of *focal* tubercle. The relative pro-

portion of leucocytes and epithelioid cells in different tubercles may vary but the resultant response to first infection will always be focal. The tubercles will be very definite, isolated and discrete formations with bacilli near the centre and concentric layers of epithelioid cells interspersed with leucocytes forming the periphery. And as we look at them we think of the wall or barrier about which we have said so much. These reactions go on in a perfectly orderly manner. Microscopically they begin to be evident a few hours after inoculation if ordinary doses of bacilli are used: macroscopically they present themselves to view from about the eighth to tenth day on. Meanwhile the guinea pig or rabbit does not exhibit the least symptom of constitutional disturbance from the time the first inoculation is made until the fourth or fifth or sixth week when the disease is progressing rapidly throughout the body.

But note that what I have described applies only to the results of a *first* infection with tubercle bacilli. If I wait until these initial tubercles develop and then reinfect my animals I may get a response of a totally different character. We may watch this response very prettily if we perform our reinfections in the skin, (intracutaneously), which we can always keep under observation. We then find that there is an immediate reaction to the reintroduction of tubercle bacilli. Four or five hours after reinfection the tissue at the point of infection begins to inflame. This acute inflammation progresses and after twenty-four or forty-eight hours may be marked both in extent and intensity. It then subsides and a few days later anatomic tubercle begins to develop in the spot. But it develops differently than did the tubercles aroused by the first infection. It appears earlier and has an accelerated development and soon subsides. And unless the reinfecting dose was overwhelming the reinfection may get no further than the skin. In other words, the reaction which we now get is comparable to what we observe when we reinoculate vaccine virus into a person who not long before has had a vaccine "take" or if we reinoculate an animal that has a chancre with a fresh dose of syphilitic virus. The reaction is accelerated and abortive and partakes more of the nature of a pure inflammation, though this latter feature will depend largely on the size of the reinfecting dose.

We are here dealing with a very striking phenomenon, the importance of which is just beginning to be generally recognized. We may pause here for a moment and generalize and say: that the development of anatomic tubercle in the body, consequent upon the introduction of tubercle

bacilli, so changes the body that all its tissues react entirely differently from the normal to the application of any soluble protein products of the tubercle bacillus or to the reintroduction of living or dead tubercle bacilli.

Everyone of you has become more or less familiar with this type of reaction in your application of the various cutaneous tests to detect tuberculous infection. And if you have followed the experimental and clinical observations that have been made on this reaction you have learned: (1) That the reaction occurs only when anatomic tubercle is present somewhere in the body. (2) That the reaction begins to manifest itself with the establishment of tubercle. (3) That up to a certain point, as the tuberculous involvement or disease progresses the intensity of the reaction increases. (4) That as tuberculous disease heals the intensity of the reaction tends to become less and less. (5) That inasmuch as tubercle is perhaps never entirely eradicated from the body, the power to react is probably never completely lost. These facts are as certain and as easily demonstrable as any that we know concerning tuberculosis. When we formulate them we are not indulging in the least bit of speculation. And we would state it as a law that the development of tubercle endows the animal body with a capacity to react against the tubercle baccilus or any of its products which it did not possess before. We would go further and state that not only the skin, but all the tissues of the body, the pleura, the peritoneum, the lungs, the liver, etc., take on this changed capacity to react. And we would also recall that no one has yet succeeded in obtaining this changed reactivity in an animal unless he first gave the animal a focus of tuberculous infection.

But with the development of anatomic tubercle other deep seated changes also take place in the animal body. For twenty years it has been recognized that an animal with tubercle can withstand relatively enormous numbers of living virulent tubercle bacilli as compared with a normal animal. For twenty years and more the details associated with this phenomenon of acquired increased resistance have been studied and the results of these studies may be summarized as follows: (1) The development of anatomic tubercle endows the body with the power to resist greatly increased numbers of tubercle bacilli. (2) This increased resistance to infection manifests itself with the establishment of the first foci. (3) Up to a certain point resistance is directly proportionate to the extent and severity of the initial disease. (4) With the healing

of the diseased foci resistance diminishes. (5) If the animal remains tuberculous the increased power to resist is probably never entirely lost, nor does resistance sink to the level which obtained before the animal was first infected. Furthermore, so far as resistance is modified by the introduction of tubercle bacilli or any of its products, this increased resistance can be obtained only by making the animal tuberculous; that is, by inoculating the animal with *living tubercle bacilli*. It is true that non-tuberculous animals may be treated with tuberculins of various types and that because of such treatment their serum may contain such bodies as agglutinins, precipitins, opsonins, etc., but such animals are not resistant to infection as compared with normal, non-tuberculous ones. We therefore assume that the ordinary serological antibodies that are met with in tuberculous infection have no direct connection with resistance to infection. In short, when we consider the effects of the tubercle bacillus and any of its products only the tuberculous animal has a relative immunity to tuberculosis.

For a number of years several of us have been studying this changed reactivity (hypersensitiveness, allergy) and this increased resistance of tuberculous animals. We have never failed to be impressed by the points of contact that the two phenomena exhibit. We have therefore wondered whether there was any close or direct relationship between the two. We have thought it reasonable to speculate that perhaps resistance to infection dwelt in this capacity of the tissues of a tuberculous animal to react immediately in an inflammatory manner. It might well be that this initial acute inflammation and the subsequent accelerated development of tubercle took care of the invading bacilli in a more satisfactory manner than did the tissues under ordinary normal conditions. For here the tuberculous animal responds with exaggerated or overreaction to irritation and throws up a barrier against the bacilli much more actively than normally and before the bacilli can get under way. From the point of view of the effect of pure inflammation we had some warrant for our belief. Pawlowsky had already demonstrated that if he first inflamed a guinea pig's knee-joint with a sterile irritant such as turpentine, alcohol or quinine, staphylococci which he then introduced into the joint would not disseminate beyond it although twenty-four to forty-eight hours after the injection of staphylococci into a normal joint he could recover the microörganisms from the blood and organs of the guinea pig. Isayeff had also shown "that the peritonitis induced by a variety of sterile irritants such as a foreign blood

serum, bouillon or normal salt solution, temporarily increases resistance to subsequent intraperitoneal inoculations of bacteria." We have therefore done many experiments to validate our view. I regret that to-night I cannot bring to you absolute and definite proof that increased resistance to infection is bound up in and dependent upon overreaction or hypersensitiveness of tissue, but I think that I do not go too far in saying that all the facts point that way.

We can inoculate normal guinea pigs or rabbits subcutaneously in the groin with a good dose of a more or less virulent strain of tubercle bacilli. After several weeks we find that glandular swellings are developing in the groin and that at the same time the animals' skins are hypersensitive to tuberculin. We can now inoculate such animals intravenously with large quantities of a virulent strain and at the same time inoculate normal animals in a similar manner. The normal animals do not suffer the least inconvenience or immediate symptoms from the inoculation. But within a few hours the reinfected tuberculous animals fall ill and from within twelve hours to four days many of them may die if the dose has been large enough. Those that do not die gradually recover from their acute illness and as a rule live on for months, many of them for a year or more, never dying of tuberculosis.

If we section the animals that have fallen acutely ill and have died shortly after reinfection we find that their lungs exhibit areas of haemorrhagic exudation or acute pneumonia or oedema which we find is the result of tubercle bacilli lodging in already sensitized soil which has overreacted. Controls sectioned at the same time, up to about eight or ten days after infection, never show these acute pathological reactions. We find therefore that animals which we know have increased resistance as well as increased tissue reactivity develop symptoms of illness while they overreact, and we also find that in response to reinfection their tissues react in a particular manner as compared with what occurs after the first infection. The character of the reaction, if the number of organisms at any point is sufficiently large and if the tissues have a very high degree of hypersensitiveness, is exudative or inflammatory, resulting in areas of pneumonia or congestion. A first injection of tubercle bacilli, you will remember, never produces such an effect. It excites the body to react only with focal tuberculous change, never with a diffuse process. Remember, however, that I am speaking always of what occurs as the immediate result of the first infection. Remember that a first infection will sensitize an animal after it has pro-

gressed for from ten or fifteen days on, and that then any extension of this infection will occur in hypersensitive soil. At any rate, as a result of such experiments we would postulate that the diffuse types of tuberculous lesion, the pneumonias, the pleurisies with effusion, the hydrops of joints, can occur only as the result of reinfection and never upon first infection and that the result of first infection is always a focal, invested tuberculosis. If the reinfecting dose of bacilli is small then the lesion may also be focal but the foci develop much more rapidly than upon first infection, and they are abortive and tend to disappear.

You see therefore that, beginning with a study of resistance to tuberculosis, we are rapidly drifting into the realms of pathology and symptomatology, if we pay attention to the wealth of observations that this study of resistance serves up to us. For the first time we can attempt to explain on a solid footing the tremendous variety of lesion that the tubercle bacillus produces in the body, a pathological result that has puzzled and confused observers since autopsies were first performed. As a rule we can affirm that it is the bacillus that is the constant and the animal that is the variable. I can take the same emulsion of the same bacillus and use the same dose to inject a series of animals. If the animals are normal I always obtain the same early result, focal tuberculosis. But if the animals are already tuberculous then almost every variety of lesion is likely to occur and the type of lesion will depend on two factors: on the number of microörganisms brought to bear at any one point and the degree of hypersensitiveness or overreaction of the tissue at this point.

Again, we note that symptoms of acute illness occur only in reinfected animals and never in the controls. This suggests what is probably true, that acute tuberculosis never develops as a first infection, that it is acute because it is more of the exudative type and that its exudative type and the acute symptoms are simply expressions of resistance, of overreaction, of immunity, of hypersensitiveness, call it what you will.

I wish I had time to develop this phase of the inquiry for as a rule I find myself growing unaccustomedly eloquent in examining the relationship of resistance to the pathology and symptomatology of tuberculosis. But I have perhaps already taken too much of your time and, like the best writers of fiction, shall close the story by suggestion.

Yet I cannot close without taking this opportunity of making a plea that all, who have a lively interest in resistance to tuberculosis, to syphilis and to other infections and to cancer, study these problems from

the angle of tissue reactions to irritation. The field is tremendous and has hardly been scratched in a systematic way. We must study the biology of these pathological formations in a developmental manner from the first signs of reaction to irritation right through to ultimate loss of tissue. For many years our pathologists have studied finished tuberculosis as they encountered it at autopsy and have only too often sought to unravel our most difficult problems by meticulous observation of cavity and cheesy patch. But the slender threads that hold the solution to the riddle have long been cut down in the cavity. What we need are careful anatomical and physiological studies of the relation of tubercle to its environment; tubercle of all kinds, tubercle in all stages of development, tubercle as a first infection and tubercle as reinfection, human tubercle in the chicken and avian tubercle in the guinea pig. What we do not know about tuberculosis almost staggers the imagination when we consider the time that has been spent on its study. How little we really and actually know about what happens after the bacillus has passed the lips and anterior nares! I would therefore appeal for workers in the field. No one man, no school, can discover it all, but if we work together then perhaps we may partially succeed in satisfying that intense longing which all of us have:—really to KNOW a little corner of our chosen study before we die.

GROUP STUDIES IN TUBERCULOSIS UNDER DIFFERENT CLIMATIC CONDITIONS[1]

WALTER C. KLOTZ

From the Barlow Sanatorium, Los Angeles, California

The various claims that have been made as to the benefits of climate in tuberculosis rest largely upon personal impression and, in many instances, upon local enthusiasm. This was strikingly brought out several years ago during a discussion of the subject before a meeting of the National Association for the Study and Prevention of Tuberculosis (1). There is a want of comparative statistics based upon records of cases. A number of the Eastern sanatoria have for years issued annual reports containing tabulations of cases treated and discharged. But, with the exception of the tuberculosis sanatoria maintained by the Federal Government, the only sanatorium west of the Mississippi Basin that issues such reports regularly is the Barlow Sanatorium of Los Angeles, California. Such reports, covering the results obtained at the well known health resorts of the Southwest, if compiled systematically and according to a standard scheme of classification, would be especially interesting. But, even if they did exist, a comparative study of such statistical material would still have the disadvantage that it had been compiled by different observers and would be subject to certain differences of interpretation of cases, and of classification of their condition on admission and discharge. The value of such a study would depend necessarily upon a careful correlation of similar groups or classes of cases.

It has been my own unique fortune, however, to have served as resident medical officer of two tuberculosis sanatoria enjoying widely different climatic conditions: one located near the Atlantic seaboard; the other on the Pacific coast. These were the Vermont Sanatorium and the Barlow Sanatorium of Southern California. Both of these institutions belong to the class of privately endowed, semi-charitable sanatoria. Each of them receives patients from about the same walks of life who follow similar occupations and represent about the same percentage of races and nationalities.

[1] Read before a meeting of the Los Angeles County Medical Association, May 4, 1916.

Since the results at each institution were obtained during my residence and under my own supervision and are, consequently, more or less a criterion of my own work, the personal factor in each instance ought to be the same. It would seem possible to institute a comparison free from bias and based upon intimate first-hand knowledge of the results obtained in two localities dissimilar in climate. Such a comparison not only would be free from the objection of native prejudice and enthusiasm, but would also have the advantage of a singleness of viewpoint not ordinarily possible in surveying statistics from different sources. The study of the rôle of climate in disease is particularly barren of just this sort of comparison, which, though empirical, possesses the value of practical significance.

Let me emphasize at the outset that I am not seeking to compare two institutions but two climatic regions. The methods of treatment in each institution were essentially the same, that is, general hygienic regimen, with the use of tuberculin in about the same percentage of selected cases. Where tuberculin was employed, the same preparation from the same source was used and the indications and dosage were essentially the same in each instance. Lung compression was not used in any of the cases. The length of stay was longer in the Barlow group and for that reason exercise and graduated labor could be carried out more extensively than in the Vermont group. These two differences may have been important factors and will be discussed again more fully below. As far as observation and reëxamination after discharge are concerned the advantage lies also with the California group of patients. Their homes were usually at some distance and their examinations were necessarily less frequent.

As all other conditions were so nearly alike, it would seem that a comparative study of the results obtained in each institutuon would offer an index as to the part played by climate in the treatment of these two groups of cases. It will be of interest, therefore, before proceeding to a study of the clinical features, to consider briefly the climatic conditions of the two localities.

The Vermont Sanatorium is located near Pittsford, a small town eight miles north of Rutland, on the western slope of the Green Mountains, at an altitude of 600 feet. The sanatorium buildings, of exceptionally substantial construction, admirably planned and equipped for the purpose, are located upon a fan-shaped moraine or shelf of gravel and sand that slopes off steeply at all sides except for a narrow neck of land

at one end, thus providing ideal conditions for drainage. The site is protected by a grove of pine and hemlock and by higher hills toward the north and northwest. The climate is typical of New England, extremes of temperature, short hot summers, long cold winters, subject to marked sudden variations, considerable rain and snow, moderately high humidity, but protected from the influence of the severe Atlantic storms by the backbone of higher mountains toward the east.

The Barlow Sanatorium is situated within the limits of the city of Los Angeles at an altitude of about 300 feet. The Sanatorium buildings consist largely of small open-air cottages, are located upon the sides and floor of a ravine or cañon and are surrounded by some eight hundred acres of park reservation, much of which is wooded with eucalyptus, pine and cedar. The hill forming the slopes of the cañon is composed of a crumbly limestone shale overlaid with a loamy soil, while the floor of the cañon consists of a layer of heavy loam overlying a stratum of fine sand. The situation is well protected from winds and in summer is rendered cooler by a draught of air through the cañon. The climate of Los Angeles is typical of Southern California; absence of extremes of temperature with, however, marked daily fluctuations, insuring cold nights throughout the year. There is a moderate rainfall occurring mostly within a short period of the year, moderate humidity, occasional frost but no snow, and a large percentage of possible sunshine. Electrical storms are rare. The differences in climate of the two localities are shown more clearly by the graphic charts (see Charts 1 and 2), compiled from the United States Weather Reports from data furnished through the courtesy of the Chief of the Weather Bureau, Charles F. Marvin of Washington, and with the assistance of the meteorologist of the Los Angeles Weather Station, Dr. Ford Carpenter, to whom I am indebted for many valuable suggestions in the preparation of this work.

In order to make these charts applicable to the present study, the curves for Los Angeles represent the average for the years 1913, 1914 and 1915, the period during which the Barlow group of patients was under observation; while the curves for Vermont represent the average for the years 1909, 1910 and 1911, the corresponding period for the Vermont group of patients. As there was no official weather station at either Pittsford or Rutland, the reports used were those from Wells, Northfield and Burlington. the nearest stations to Pittsford for which records are available. The names of the observation stations are noted at the beginning of each curve.

From a study of these weather curves the advantage as far as climate is concerned would certainly appear to be with Los Angeles. The principal factors in its favor are the greater percentage of sunshine and the more equable temperature for the whole year, with sufficient daily varia-

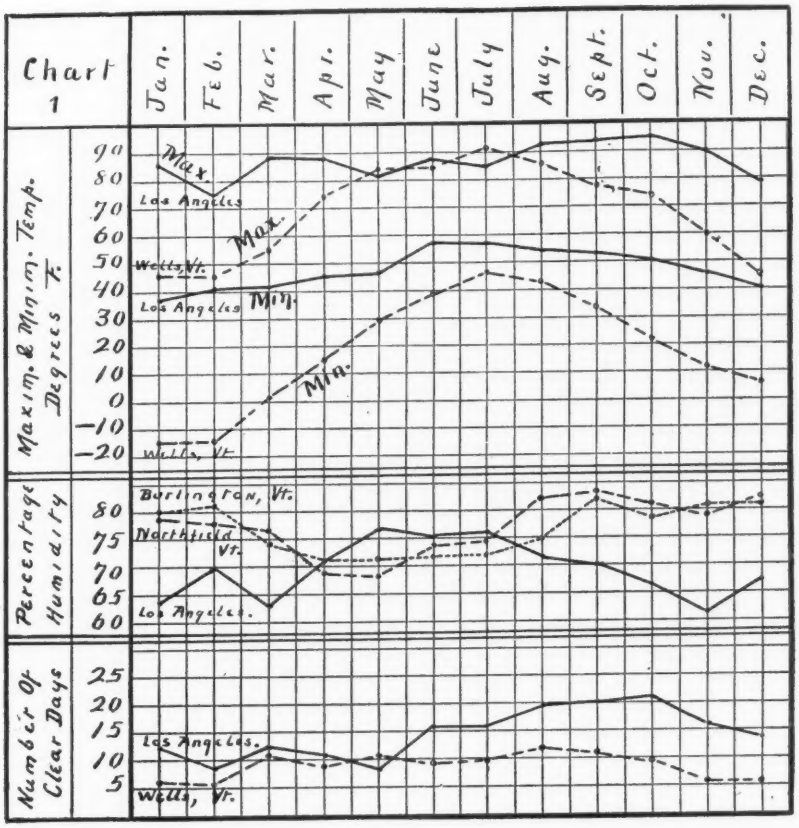

CHART 1. MAXIMUM AND MINIMUM TEMPERATURE, NUMBER OF CLEAR DAYS AND HUMIDITY, CALIFORNIA AND VERMONT

The figures for Los Angeles represent the average for the years 1913, 1914 and 1915. The figures for Vermont represent the average for the years 1909, 1910 and 1911. Wells, Burlington and Northfield were the stations nearest stations to Pittsford for which weather reports were available.

tion to insure cool nights in summer. There is a long period each year without storms or rain, permitting the greatest amount of time out of doors with the least amount of discomfort. In fact, patients at the

sanatorium are literally living out of doors all the time, as the open air cottages have no means of being closed on two of the sides except by means of protecting awnings. The cottages, moreover, are not capable

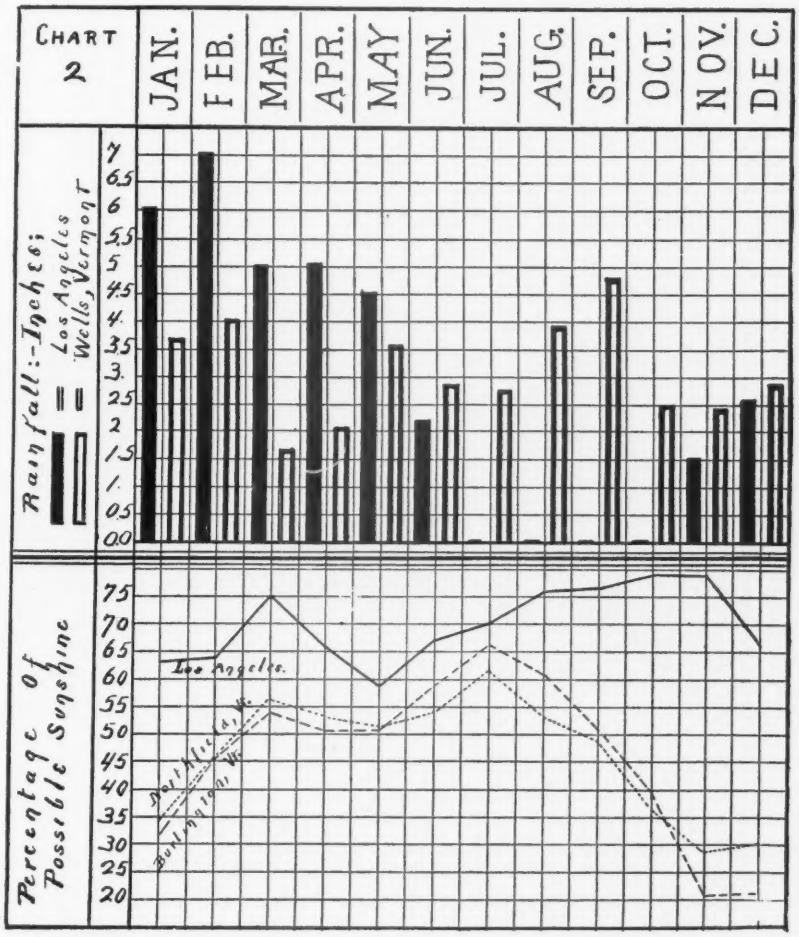

CHART 2. RAINFALL AND PERCENTAGE OF POSSIBLE SUNSHINE, CALIFORNIA AND VERMONT

The figures for Los Angeles represent the average for the years 1913, 1914 and 1915. The figures for Vermont represent the average for the years 1909, 1910 and 1911. Northfield and Burlington were the stations nearest Pittsford, Vermont, for which weather reports were available.

of being heated, so that there is an absence of contrast between in-doors and out-of-doors and an absence of the chilling encountered when leaving steam heated buildings in cool weather. During the short season

when the rains are frequent, the downpour may be torrential, making it impossible to venture out. There is usually considerable fog during the spring months, April, May and June, the period registering the highest humidity (see Chart 1). The prolonged hot, dry season, lasting well into October or even November, often becomes monotonous to some patients, especially those from the East who miss the tonic, invigorating effect of the dry, cold winters of New England and the Adirondacks.

In studying the results of treatment the number of cases in each group was limited to 200 cases in order to include only patients that had been treated and observed by myself for some time before discharge. Each group, however, comprises 200 consecutive, unselected cases taken in order of their discharge, omitting only such cases whose sanatorium residence was less than thirty days and who were considered technically "untreated."

The cases for each class are tabulated according to the condition on admission and discharge. The traced cases for each class are, moreover, tabulated according to their condition from one to two and one-half years after discharge. It has been generally accepted that the benefits of sanatorium treatment can not be estimated by the inmediate results, as shown by the condition of the patient when discharged, bur rather by the "after results" of treatment as shown by the condition of patients when they have returned to their former surroundings or resumed some regular occupation. In these tables the term "Well" is used to indicate that the patient was working or able to work; while "Living" means that the patient was still taking the "Cure." "Living" does not include "Well."

In compiling the results of the Vermont group I was assisted largely by notes I had preserved from the statistical study of 350 cases treated at the Vermont Sanatorium (2). For additional data I am indebted to Dr. E. F. Rogers, the present medical director and superintendent of the Vermont Sanatorium, who was kind enough to look up the records of some fifteen cases.

The results of these studies are shown in tables 1 and 2, according to which the findings for each class were decidedly in favor of the Barlow group. This applies not only to the immediate results on discharge but also to the ultimate or "after results" of treatment. These are more clearly shown by the graphic chart headed, "Summary of after results" (Chart 3), in which the different classes of cases are more directly compared. A study of this graphic chart shows that in each class of case

TABLE 1

Immediate results: 200 patients discharged from the Vermont Sanatorium, March 1, 1909, to March 1, 1911

CLASSIFICATION ON ADMISSION		CONDITION ON DISCHARGE				
		Apparently arrested	Quiescent	Improved	Failed	Died
Incipient	69 = 34.5%	38 = 55.0%	23 = 33.3%	8 = 11.0%	0	0
Moderately advanced, I	40 = 20.0%	7 = 17.5%	12 = 30.0%	15 = 37.5%	6 = 15.0%	0
Moderately advanced, II	70 = 35.0%	5 = 7.17%	22 = 31.4%	25 = 35.7%	15 = 21.4%	3 = 4.3%
Moderately advanced, III	17 = 8.5%	0	3 = 17.6%	6 = 35.2%	6 = 35.2%	2 = 11.7%
Far advanced, III	4 = 2.0%	0	0	2 = 50.0%	2 = 50.0%	0
	200 = 100.0%	50 = 25.0%	60 = 30.0%	56 = 28.0%	29 = 14.5%	5 = 2.5%

After results of 141 traced patients from above group, condition from one to two and one-half years after discharge, from the Vermont Sanatorium (including those who died at the Sanatorium)

CLASSIFICATION ON ADMISSION		CONDITION ON DISCHARGE																							
		Apparently arrested						Quiescent						Improved						Failed				Died	
		Well		Living		Dead		Well		Living		Dead		Well		Living		Dead		Well		Living		Dead		Dead	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Incipient	46 = 32.6%	16	66	4	16.6	4	16.6	12	66	3	16.6	3	16.6	3	75.0	0		1	25	0		0		0		0	
Moderately advanced, I	29 = 20.0%	4	80	0		1	20.0	6	60	3	30.0	1	10.0	2	22.0	6	66.6	1	11	1	20.0	0		4	80	0	
Moderately advanced, II	50 = 35.4%	1	50	1	50.0	0		7	43	3	18.7	6	37.5	6	27.2	6	27.2	10	45	0		3	42	4	57	3	100
Moderately advanced, III	14 = 9.9%	0		0		0		0		1	33.0	2	66.6	0		2	40.0	3	60	0		0		4	100	2	100
Far advanced, III	2 = 1.4%	0		0		0		0		0		0		0		0		0		0		0		2	100	0	
Totals	141 = 100.0%	21	67	5	16.0	5	16.0	25	53	10	21.0	12	25.0	11	27.5	14	35.0	15	37	1	5.5	3	16	14	77	5	100

TABLE 2

Immediate results in 200 cases discharged from the Barlow Sanatorium, between March 1, 1913, and January 1, 1916

CLASSIFICATION ON ADMISSION		CONDITION ON DISCHARGE				
		Apparently arrested	Quiescent	Improved	Failed	Died
Incipient	9 = 4.5%	6 = 66.6%	2 = 22.2%	1 = 11.1%	0	0
Moderately advanced, I	28 = 14.0%	20 = 71.4%	5 = 17.8%	2 = 7.1%		1 = 3.5%
Moderately advanced, II	124 = 62.0%	43 = 34.6%	43 = 34.6%	25 = 20.0%	10 = 8.0%	3 = 2.4%
Moderately advanced, III	32 = 16.0%	0	4 = 12.5%	13 = 40.6%	10 = 31.2%	5 = 15.6%
Far advanced, III	7 = 3.5%	0	0	2 = 28.5%	2 = 28.5%	3 = 42.8%
Totals	200	69 = 34.5%	54 = 27.0%	63 = 31.5%	22 = 11.0%	12 = 6.0%

"After Results" in 117 traced patients from above group; condition from one to two and one-half years after discharge

CONDITION ON ADMISSION		CONDITION ON DISCHARGE																							
		Apparently arrested						Quiescent						Improved				Failed						Died	
		Well		Living		Dead		Well		Living		Dead		Well		Living		Dead		Well		Living		Dead	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Incipient	4 = 3.4%	3	100											1	100	0		0		0		0		0	
Moderately advanced, I	20 = 17.0%	14	93			1	6.7	2	100					2	100	0		0		0		0		2	100
Moderately advanced, II	66 = 56.4%	28	96	1	3.5			16	66	7	29	1	7.0	1	10	7	70	2	20	0		0		6	100
Moderately advanced, III	21 = 17.9%	0		0		0		1	50	1	50	0		1	11	6	66	2	22	0		2	40	3	60	10	100
Far advanced, III	6 = 5.12%	0		0		0		0		0		0		0		2	100	0		0		1	50	1	50	4	100
Totals	117 = 100.0%	45	95	1	2.0	1	2.1	19	68	8	28	1	3.6	5	20	15	62.5	4	16	0		3	43	4	57	22	100

the results are better for the Barlow group than for the Vermont group and consequently also for the Barlow group as a whole.

If we come to analyze this chart more carefully, however, we can discover several sources of error which may have made the results for the Vermont group appear less favorable than they actually were. Taking the "Incipient" class, for example, we find on referring to tables 1 and 2 that this class constituted 34.5 per cent of the cases of the Vermont

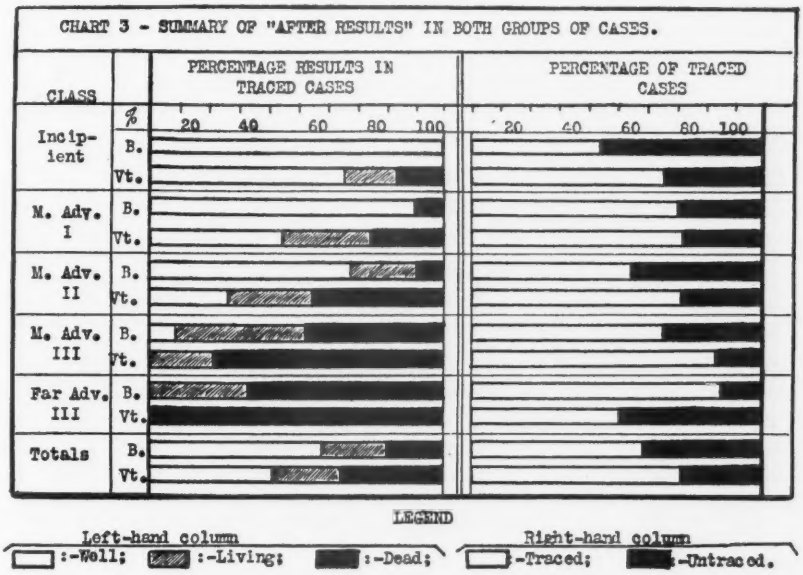

CHART 3. SUMMARY OF "AFTER RESULTS" OF TREATMENT

Condition one to two and a half years after discharge. Left hand column shows results for both groups of cases compared according to stages of disease (condition on admission) and also for total number of cases in each group. Right hand column shows percentage of traced cases for each group arranged similarly.

NOTE. "Well" means working or able to work—*clinically well.* "Living" does not include "well" and means still unable to work on account of illness, i.e., still following treatment.

group, but only 4.5 per cent of the Barlow group. One reason for this marked relative difference is the fact that we do not classify as "Incipient" as frequently as we formerly did. With our present ideas as to childhood infection and long period of latency in pulmonary tuberculosis, the term "Incipient" becomes almost a hypothetical conception. Many of the cases of the Vermont group classified at that time as "Incipient" would, accordingly, be classified now as "Moderately Advanced

I," or, possibly, "Moderately Advanced II." Such a rearrangement of cases, throwing some, at least, of the "Incipient" cases of the Vermont group into the "Moderately Advanced" class, would naturally tend to make results for this class show up more favorably. Furthermore, it is only to be expected that anyone applying himself to a certain line of study and work, will attain greater proficiency and skill and, in the course of years, become more critical in judging results. Not only will the findings of physical examinations become more complete but their interpretation becomes more exact. Changes will be noted that were formerly overlooked. This is especially true in connection with signs of cavity where a positive diagnosis will be made more often even in the absence of classical text-book signs. This is particularly true if the physical findings have been repeatedly confirmed by anatomical changes at autopsy. Accordingly, therefore, it would follow that many of the cases of the Vermont group, aside from those in the incipient class, would be thrown into the more advanced class if studied at the present time, all of which would tend to work out in favor of the Vermont group of cases. To obtain a true and accurate correlation of two groups of cases, even by the same individual, would require that they be studied simultaneously. This is a condition which it is naturally impossible to fulfill.

A further feature that would help to make the results of the Barlow group appear more favorable is the fact that many of the cases of this group represent more the chronic fibroid type with acute exacerbations. These are the cases in which anatomical changes, as shown by resonance as well as by voice and breath changes, are more or less extensive, but in which the signs of active inflammation, congestion and softening are heard only over a relatively small area or are not intense in character. In other words, we do not find in the Barlow group as many acute ulcerative forms as in the Vermont group. This fact is borne out also by a relative infrequency of haemoptysis.

As far as prominent clinical features are concerned it is interesting to note that in 64.5 per cent of the Vermont group of patients, bacilli were found in the sputum at some time during residence, while of the Barlow group 56 per cent gave positive sputum examinations. The temperature was 100° F. or over in 16 per cent of the Vermont group on admission and in 11.5 per cent on discharge; while in the Barlow group the temperature was 100° F. or over in 6.5 per cent on admission and in 3 per cent on discharge.

No careful study has been made of the dietary at the two institutions upon a calory basis. In a general way, however, the dietary was more excessive, more costly and included a greater percentage of protein at the Vermont Sanatorium. The average immediate gain in weight was also greater in the Vermont group than in the California group. The differences in results in the two groups cannot be laid, therefore, to difference in care of patients. The cost of maintenance per patient per week was considerably higher in Vermont than in California. While not necessarily a criterion, owing to difference in construction, location, cost of heating, etc., the Vermont patients were at least cared for as well as the California patients.

The question may be raised, very properly, whether conditions after discharge were as favorable in the case of the Vermont patients as in those treated in Southern California, and whether the occupations followed by the patients in the first group were more hazardous from the tuberculosis point of view and whether the living conditions were as good as among the second group. It might be argued that this factor may have been an important one in causing a greater proportion of relapses among the Vermont patients. To determine the exact conditions after discharge without more detailed data is of course difficult. In neither group were the patients followed up by personal visits or reported upon by visiting nurses or social workers. We were dependent upon correspondence, written reports, or in some cases statements by a physician or even casual information furnished by other patients.

As a rule, patients resume their former occupation or some similar occupation, after discharge. The study of occupations at the time of admission, therefore, might offer some basis for a study of occupational and living conditions of the two groups. A survey of the accompanying tables of occupations shows with few exceptions a striking similarity of figures.

The larger groups, such as office workers, professional trades, commercial pursuits and house workers are very close. Among the Vermont patients were a number of granite cutters comprising 15.8 per cent of all the men, while no granite cutters were included in the California group. Furthermore, there were, among the Vermont patients, 15.2 per cent classified as farmers, while of the Barlow group only 4 per cent were classified as farmers and ranchers. These two pursuits, granite cutting and farming, are, probably, the most unfavorable, from the point of view of tuberculosis and these alone constitute 31 per cent of

the men in the Vermont group. In this group of patients, therefore, we certainly would find certain factors favoring relapse, not only on account of conditions in connection with the occupation but, in the case of farmers, on account of conditions of living.

Tables showing percentage of different occupations in Vermont and Barlow groups of patients

MEN	VERMONT	BARLOW
	per cent	*per cent*
Granite cutters	15.8	0.0
Office workers	15.2	38.0
Farmers	11.9	4.0
Students	11.2	7.0
Commercial pursuits	8.6	13.0
Drivers and teamsters	6.6	5.0
Factory operatives	2.6	3.0
Marble workers	2.6	0.0
Slate workers	1.3	0.0
Surveyors	0.0	1.0
Teachers	1.3	3.0
Lawyers	0.6	1.0
Dentists	0.6	0.0
Physicians	0.0	2.0
No occupation	0.6	0.0
Miscellaneous trades	20.0	21.0
Musicians and actors	0.0	2.0

WOMEN	VERMONT	BARLOW
	per cent	*per cent*
Houseworkers	54.7	51.7
Office workers	13.5	18.0
Students	12.5	8.6
Teachers	7.5	6.8
No occupation	4.5	4.2
Needle women	4.0	1.7
Factory operatives	1.5	1.7
Trained nurses	1.0	1.7
Musicians and actresses	0.5	0.8
Students and practical nurses	0.0	4.2

Among the women patients the figures for various occupations are more alike. In neither group of patients was there any considerable number of factory operatives or shop workers, who are usually considered especially predisposed to tuberculosis. House workers constitute over 50 per cent in each group. As was noted above, however, it is difficult,

from a simple tabulation of occupations, to determine definitely the standards of living and the occupational surroundings after discharge. The general impression would be, however, that the standard of living for the California group was probably higher, especially in respect to housing. Under existing climatic conditions it is naturally easier for them to obtain a greater amount of fresh air in their living and sleeping quarters. On the other hand, conditions in offices and shops as far as ventilation is concerned are probably no better in California than Vermont. In the case of housewives and houseworkers the many difficulties and trials are probably as numerous among the California patients as in Vermont. The domestic help problem is fully as difficult; that is, workers are equally scarce while wages are higher.

As far as personal habits of patients after discharge are concerned it is probably even more difficult to arrive at any definite conclusion than in the case of occupational or living surroundings. The same is true of indulgence in alcohol. There probably is more tendency to pleasurable pastimes, frequently involving late hours, in California than there would be in Vermont, while indulgence in alcohol would be more in the way of moderate social drinking. Among the Vermont patients indulgence in alcohol would be more apt to be periodic but more excessive if taken at all. A further study of predisposing factors would show no difference between the two groups. Of the Vermont group all were residents of the United States and most of them were residents of Vermont; that is, 70 per cent. Of the Barlow group 9 per cent were residents of California and 71 per cent residents of the United States other than California. The distribution as to sex and age was so similar as to make special tables superfluous. The incidence of tuberculosis in the family and association with the disease were about the same in the two groups.

IMPRESSION

My impression as placed against statistics would have looked for less difference in results. On the other hand, in recalling the work in Vermont it would certainly seem that haemoptyses occurred much more frequently than in California, especially during the spring and fall of the year, and that they were more profuse, more persistent, less easily controlled, more frequently followed by extension, and consequently more dreaded. Elevation of temperature was also more frequent and more persistent, while cavity cases did not do well. Moreover, there

was in California an absence of the epidemics of acute catarrhal infections that accompanied the spring thaws and played such havoc with our patients in Vermont.

As the differences in results in the two groups were so marked, they were checked off against tabulated statistics of "After results" of treatment taken from the annual reports of both institutions, covering eleven years from the Barlow Sanatorium and six years from the Vermont Sanatorium. These tabulations based upon work done in part by my predecessors at the Barlow Sanatorium and my successors at the Vermont Sanatorium showed very nearly the same results as those brought out in the two groups previously considered.

Before accepting the more favorable results in the Barlow group as being due to climatic differences, it will be necessary to consider carefully two factors that may have an important bearing upon these results. One of these is the fact that the average length of stay among the Vermont group was only 4.6 months as against 7.4 months for the Barlow group. This difference in length of treatment is probably the more significant factor. The fact, also, that graduated labor and exercise were applied less generally and less systematically among the patients of the Vermont group may also have considerable bearing upon the subject, in so far as during the shorter say there was less or no opportunity for hardening them and protecting them against recurrence. Just as soon as they were in condition to do a little work they would leave and return to their former surroundings. On the other hand, it must be mentioned that among the Barlow group more of the patients were living in California, away from home, and dependent upon their own resources for a livelihood so that they were unable to enjoy the protection of a home when they left the sanatorium. In the case of some of the Vermont patients it must however be conceded that "going home" frequently meant going back to the same unfavorable surroundings of hard living, inadequate dietary and poorly ventilated dwellings that had predisposed them to their illness.

Granted, moreover, that the better results obtained in California may be ascribed in part, at least, to more favorable climatic conditions, it must be pointed out that these results were obtained only through carefully regulated sanatorium regimen, prolonged in some cases over several years. It must also be borne in mind that there were a number of patients in the Barlow group who had taken the cure elsewhere before coming to California—some in other sanatoria, perhaps for too short a

time—who, having learned their lesson through sad experience, were more inclined to follow the treatment seriously and earnestly.

SUMMARY

Two equal groups of cases, representing the same races and nationalities, following similar occupations, taken from similar conditions of life, were treated in two similar institutions by the same individual and by the same methods. The records and statistics of these two groups of cases were also compiled by the same individual.

These two institutions were located in different parts of the country enjoying widely different climatic conditions, one in Vermont and the other in Southern California.

The results obtained in California were distinctly better than those obtained in Vermont. This refers not only to the immediate results, but also to the ultimate results after discharge.

One source of error must be considered in accepting these statistics. Although they were compiled by one individual, it would seem probable that the cases in the California group, having been studied more recently and after greater experience, were viewed more critically; that, consequently, these cases were classified as more advanced than they would have been had they been studied simultaneously with those of the Vermont group.

It was also shown that elevation of temperature occurred less frequently and subsided more rapidly in California than in Vermont; that haemoptysis was less frequent, less persistent and less profuse; and that intercurrent catarrhal infections were less common in California.

The dietary was more generous in Vermont than in California not only as to total quantity, but also as to the proportion of protein. The average gain in weight was also greater and more rapid in Vermont than in California.

The average length of stay was considerably shorter in Vermont than in California and systematized exercise and graduated labor were carried out less extensively in the Vermont group than in the California group.

CONCLUSIONS

After considering carefully all collateral factors and eliminating possible sources of error, the differences in results obtained in these two groups of patients are still sufficiently marked to warrant the conclu-

sion that, given similar modes of living and methods of treatment, greater and more lasting benefit would be obtained under the climatic conditions of California than under those of New England.

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THE PROGNOSIS OF PULMONARY TUBERCULOSIS IN INFANCY

T. C. HEMPELMANN

From the Department of Pediatrics, Washington University, and the Saint Louis Children's Hospital, St. Louis, Missouri

The impression that tuberculosis in infancy, especially during the first year of life is almost invariably fatal, is so nearly universal as occasionally to constitute a serious menace to an already heavily afflicted child. A feeling of resignation is apt to arise when dealing with an apparently hopeless disease which bodes ill for further therapeutic endeavor. Actual studies of the fate of children infected with tuberculosis in infancy are extremely few, and it would seem to be of the utmost importance that more such studies be undertaken in order to present definite evidence bearing on this important question of prognosis.

That infection with the tubercle bacillus in childhood does not necessarily produce fatal or even serious illness in every case, is shown by the great number of children who give positive tuberculin reactions but show no other evidence of infection; that is, the disease apparently remains latent. Tuberculous infections in infants, however, must always be regarded as *active* tuberculosis, first, because such lesions in infants have probably not had time to become sufficiently walled off to insure their remaining latent, and secondly, because at this period of life the human organism is least resistant to the tubercle bacillus and infection is often followed by rapid involvement of other organs far removed from the original site of infection. Recognition of this latter fact is undoubtedly responsible for the prevailing pessimism regarding the outcome when tuberculosis affects children in the first two years of life. For example, one of the recent studies of tuberculosis in infancy contains the following:

> The tendency of tuberculosis in young subjects is toward generalization, rather than toward localization and healing. Tuberculosis is a progressive, and not a retrogressive, process in infants; consequently tuberculous infection in early infancy leads to the death of the infant with comparative rapidity.

And again:

The younger infants tend to be overwhelmed by the tubercle bacillus and no immunity develops. In children over two years, there is an increasing tendency toward immunity and consequent limitation of the lesions for a time (1).

Evidence is however accumulating which shows that a certain number of cases of true pulmonary tuberculosis, even in the first year of life, do recover, and it is very probable that this percentage can be increased if we expend as much effort and care on them as we do on those cases occurring later in life. One of the most frequently quoted studies bearing on this point is that of Pollak of Vienna (2). Pollak's figures, which are reproduced in one of the tables which follow, lose much of their value because of the unique way in which he has computed the mortality. He presents no data to show how long his cases have been followed, but merely eliminates from consideration those babies who are still under one year of age and includes in his figures those infants who are known to have died, or are in the second year of life (twelve to twenty-four months). That is, all infants who are past the first year of life are classed with the "living" although the diagnosis of tuberculosis may have been made just a month or two previous, and the disease may be running an average course to a fatal termination. He also includes among the living, two children who died of diseases other than tuberculosis. This method of classification gives figures of very questionable value for the "living" group and hence we have included in the group classed as living only those infants who have lived *at least one year* after the diagnosis was made, and were living when last heard from. For purposes of comparison, a table is presented showing our figures computed according to Pollak's method. It will be seen that the figures agree quite closely for the first year of life although they differ somewhat when each half year is considered separately.

The material for the present study consists of one hundred and thirty cases of pulmonary tuberculosis in the first two years of life, observed in the Pediatrics Services of the St. Louis Children's Hospital and Washington University Dispensary. The diagnosis was based on the presence of symptoms, pathological physical findings, positive tuberculin reaction and positive Roentgenographic findings, the presence of all four being a necessary requisite for admission to the series. The only exceptions to this were those cases in which the infants died and

the diagnosis was confirmed at autopsy. Sputum examinations were made in many instances, but not in all.

Table 1 shows the number of children who are known to have died, classified by age of decedent at the time the diagnosis was made and the interval elapsing between this time and the time of death. There were sixty-four cases in all, of which thirty-seven were under one year of age at the time of diagnosis, and twenty-seven were in the second year of life. Of the total number, forty-six infants (72 per cent) died within three months of the time of diagnosis. As many of these babies were brought to the hospital only when practically moribund, or at least in the very last stages of tuberculosis, these figures give no reliable indication of the rate of progression of the disease. Eighteen babies lived at least three months after diagnosis, nine lived over six months, seven

TABLE 1

Dead. Time elapsing between diagnosis and death

AGE	NUMBER	DIED WITHIN 3 MONTHS	LIVING MORE THAN 3 MONTHS AFTER DIAGNOSIS	LIVING MORE THAN 6 MONTHS AFTER DIAGNOSIS	LIVING MORE THAN 9 MONTHS AFTER DIAGNOSIS	LIVING MORE THAN 1 YEAR AFTER DIAGNOSIS	LIVING MORE THAN 1½ YEARS AFTER DIAGNOSIS
months							
0– 6	8	6	2	1	1	1	0
6–12	29	22	7	3	2	1	0
12–18	10	5	5	1	1	0	0
18–24	17	13	4	4	3	1	1
Total.....	64	46	18	9	7	3	1

lived over nine months, three lived more than a year, and one eighteen months old baby lived a year and a half after diagnosis and then, his tuberculosis having given no symptoms for some months, succumbed to measles and broncho-pneumonia. Of the other two who lived for over a year, one was a colored baby who when first seen at eight months of age, had a dactylitis, positive lung findings, large spleen, and numerous skin tuberculides. The Roentgenogram and von Pirquet reaction confirmed the diagnosis. The baby did very well for a short time, gaining in weight and looking much better, but then the condition slowly became worse and the baby finally died of a tuberculous meningitis a little over a year after first being seen. The other child was brought to the hospital when four months old because of a marked expiratory stridor. He had in addition definite lung findings, large liver and spleen, positive tuberculin reaction and Roentgenographic findings.

He had numerous attacks of choking and cyanosis, which repeatedly recurred up to the time of his death, one year later. The autopsy showed a miliary tuberculosis of the lungs, liver and spleen, with a large mass of caseous tracheo-bronchial glands and a moderately enlarged thymus. Four other children lived nine months or more. One of these (fourteen months old) died of scarlet fever nephritis nine months after tuberculosis was first diagnosed. The autopsy showed, in addition to the nephritis and general anasarca, tuberculosis of the lungs, tracheobronchial glands and right os calcis. Another child with definite pulmonary tuberculosis was lost sight of for some months, until its record was found among the records at the Bureau of Vital Statistics, according to which she had died of tuberculous meningitis nine months after first coming to the Dispensary. The third child (twenty-three months when first seen) showed tuberculosis of the lungs, hip, ankle and elbow, and died of meningitis not quite a year after diagnosis. The fourth child had hereditary syphilis with positive Wassermann reaction, positive von Pirquet test, tuberculosis of the lungs and ankle, numerous sinuses from broken down glands (pus from which produced tuberculosis in a guinea pig) and immense numbers of papulo-necrotic tuberculides with lichen scrofulosorum. Of the other fatal cases, the only one which merits special mention is a baby, twelve months old when first seen, who died of a suppurative pericarditis five months after the diagnosis of tuberculosis had been made, both diagnoses being confirmed at autopsy. It is thus seen that tuberculosis was not the immediate cause of death in all of the sixty-four fatal cases. What part the tuberculous infection played in lowering the general resistance can only be a matter of conjecture.

In table 2 will be found the children, sixty-five in number, who were living at the time of the last note on the history. Their ages at the time of the diagnosis of tuberculosis, and the length of time under observation (time from diagnosis to last note) are shown in the table. It will be seen that thirty-one were followed less than six months, thirty-five more than six months and thirty were observed one year or longer. It was possible to follow nineteen of these for two years or more, ten for three years or more, five for four years or more, and two for more than five years. Of the thirty infants who were observed a year or more after diagnosis, only two showed symptoms referable to the lungs, while seven showed other evidences of tuberculosis when last seen (three had tuberculosis of the spine, one involvement of the hip,

one of the ankle, one a dactylitis and one an active phlyctenular keratitis). Twenty-two of the thirty babies observed a year or longer had, at some time during this period, other manifestations of tuberculosis than those referable to the lungs and tracheo-bronchial glands. In this connection, it should be mentioned that for the purposes of this study, phlyctenular disease has been considered a manifestation of tuberculosis. Of the five babies observed more than six months, but less that a year, only one showed symptoms at the time of the last note. Of the thirty observed one year or more, ten were under one year of age when the diagnosis was made, only one shows active lung signs at present, and one other has a tuberculosis of the spine which was operated on some months ago, there having been no lung symptoms for about two years. The other twenty babies were between the ages of

TABLE 2

Period of observation of the sixty-six children living at last note

AGE	NUMBER	LESS THAN 6 MONTHS	MORE THAN 6 MONTHS	MORE THAN 1 YEAR	MORE THAN 2 YEARS	MORE THAN 3 YEARS	MORE THAN 4 YEARS	MORE THAN 5 YEARS	LUNG SYMPTOMS OF THOSE OBSERVED 1 YEAR, ++	OTHER SYMPTOMS OF TUBERCULOSIS OF THOSE OBSERVED 1 YEAR, ++
months										
0– 6	5	3	2	2	1	1	0	0	0	0
6–12	20	9	11	8	6	3	3	2	1	1 (spine)
12–18	17	8	9	9	8	4	1	0	0	2 (hip, spine)
18–24	24	11	13	11	4	2	1	0	1	4 (spine, ankle, finger, phlyctenular keratitis)
Total.....	66	31	35	30	19	10	5	2	2	7

twelve and twenty-four months when the diagnosis was first made, and only one shows lung signs at the present time, although six still show other evidences of tuberculous disease, as noted above.

The youngest baby (K. M. No. 5649) was six weeks old at the time of diagnosis. He had a cough, fever, dulness and bronchial breathing over an area in the right upper lobe, and gave a strongly positive von Pirquet reaction. The Roentgenogram showed a shadow corresponding to the area of dulness, with enlarged glands. There was a large spleen and marked pallor. The cough lasted for ten months and showed several recurrences after this time, the last one occurring twenty-two months after he was first seen. The dulness persisted seven months. This baby has been observed a little over three years, and he is now quite fat and healthy looking. He has not coughed for

over a year and has never had any other manifestation of tuberculosis, except that his spleen is still palpable. He was watched most carefully during his entire illness, and, measured by all clinical standards, there seems to be no reasonable doubt but that the condition was pulmonary tuberculosis.

Another child (P. T. No. 15963) was five months old when brought to the Dispensary because of a very pronounced expiratory stridor and cough. She had a positive tuberculin reaction, Roentgenographic and lung findings, and is living today, fifteen months after the diagnosis was first made, well nourished, healthy looking, and with neither symptoms nor abnormal physical signs. A third child (T. W. No. 9871) was six months old when, because of the usual evidences of pulmonary tuberculosis, the sputum was examined and tubercle bacilli found. He has been observed twenty months, but still shows pulmonary symptoms, as well as a phlyctenular keratitis. A fourth baby (J. T. No. 10352) presented typical lung findings, large spleen, and tuberculous cervical glands, with numerous papulo-necrotic skin tuberculides at six months of age. Soon afterwards he developed a phlyctenular keratitis, and within a year, Pott's disease. He has now been observed three years and has shown no lung signs for about two years. Another infant of eight months (F. Z. No. 7841) presented a massive involvement of one upper lobe, which remained unchanged for about five months and then began to improve. Today, twenty-six months later, he is apparently entirely well. A sixth baby (L. L. No. 8301) eight months old when first seen is living and free of symptoms twenty-two months after an undoubted pulmonary and tracheo-bronchial gland tuberculosis. G. G. No. 3617 came to the Dispensary when eight months old, when a pulmonary and cervical gland tuberculosis was diagnosed. She has been observed five and a half years and it is only during the past year that she has been entirely free from both cough and symptoms.

A. L. No. 2807 entered the hospital when one year old, with pulmonary involvement and tubercle bacilli in the sputum. During the course of the next year, she also developed a phlyctenular keratitis and hip disease. Except for the hip condition, which is still under treatment, she is entirely well, four years and nine months after tubercle bacilli were first found in her sputum. L. T. No. 7704, admitted to the hospital when twenty-three months old, had hereditary syphilis and tuberculous involvement of the lungs and ankle with sinuses of face, and tubercle bacilli were recovered from ankle. The child is living

and doing well two years later. A. H. No. 8597 had what was diagnosed broncho-pneumonia when twenty-two months old, but while testing some Petroff culture media, tubercle bacilli were grown from this child's sputum and the true condition revealed. The patient is living and apparently well twenty months later. J. B. No. 5730 had fluid in his chest when twenty-two months old, and this fluid produced miliary tuberculosis when injected into guinea pigs. This baby also developed phlyctenular keratitis later, but he is living and apparently well, with no lung symptoms, twenty months later.

It would be unnecessarily tedious repetition to enumerate all the cases, and the above will suffice to show that these were true cases of tuberculosis. There remains, however, one case to be described in some detail, because it illustrates what marvelous resistance some infants possess, not only to the tubercle bacillus, but to other infections as well. M. P. No. 5050 developed a tuberculous dactylitis and pulmonary involvement when nine months old. He had dulness and bronchial breathing which remained over seven months, and abnormal lung findings for over a year. Tuberculin and Roentgenographic findings were constantly positive. He developed multiple tuberculous abscesses in the soft parts of the leg, arms, left cheek and eye-lid, all of which left sinuses which discharged for months. He also developed cervical adenitis which broke down and discharged pus for several months. He successfully withstood in the course of his illness an attack of follicular tonsillitis, measles, double otitis media, scarlet fever and scarlet fever nephritis, and finally phlyctenular keratitis. He has been under observation for four years, has not coughed for twenty months and has had no tuberculous symptoms of any kind for over a year and a half. He is undersized, but quite fat and tanned from his outdoor life, so that were it not for the numerous scars which mark the site of the old sinuses, we would pick him out as a type of healthy, sturdy childhood.

In computing the mortality (table 3) only those cases were classed among the "living" who were observed for a period of at least one year after the diagnosis of pulmonary tuberculosis was made, leaving ninety-four cases to be studied from this standpoint. It will be seen that during the first six months the mortality was 80 per cent, and during the next six months of life, 78.3 per cent, or a general mortality of 78.7 per cent among forty-seven babies under one year of age. Among those babies who were between the ages of twelve and eighteen months, the mortality was 52.6 per cent and between the eighteenth and twenty-

TABLE 3

Mortality

AGE	NUMBER	LIVING 1 YEAR AFTER DIAGNOSIS	DEAD	MORTALITY
months				*per cent*
0– 6	10	2	8	80.0
6–12	37	8	29	78.3
0–12	47	10	37	78.7
12–18	19	9	10	52.6
18–24	28	11	17	60.7
12–24	47	20	27	57.4
0–24	94	30	64	68.0

fourth months, 60.7 per cent, making a mortality of 57.4 per cent, among forty-seven babies in the second year of life. The mortality for the two years was 68 per cent.

Table 4 is presented for comparison with Pollak's figures given in table 5, those babies being classed as living (according to Pollak's method) who have passed into the second year of life. It will be seen that the general "mortality" figures for the year correspond quite closely, although our figures are lower for the first six months of life and higher for the next six months than Pollak's. We feel, however, that a better picture of actual conditions is given if the "living infants" observed less than one year are omitted as we have done in table 3 which makes a mortality of 78.7 per cent instead of 69.8 per cent, the figure in table 4.

TABLE 4

St. Louis figures for comparison with Pollak

AGE AT DIAGNOSIS	TOTAL	FATE UNKNOWN OR STILL UNDER 1 YEAR OF AGE	1 TO 2 YEARS OLD	2 TO 3 YEARS OLD	3 TO 4 YEARS OLD	4 TO 5 YEARS OLD	5 TO 6 YEARS OLD	6 TO 7 YEARS OLD	DIED	MORTALITY
months										*per cent*
0– 6	13	3*	1	0	1	0	0	0	8	80.0
6–12	49	6*	6	4	1	1	1	1	29	67.4
0–12	62	9*	7	4	2	1	1	1	37	69.8

* Subtracted from the general figures in computing Mortality.

TABLE 5

Pollak's figures in Vienna

AGE AT DIAGNOSIS	TOTAL	FATE UNKNOWN OR STILL UNDER 1 YEAR OF AGE	1 TO 2 YEARS OLD	2 TO 3 YEARS OLD	DIED	MORTALITY
months						*per cent*
0– 6	30	9*	3	0	18	85.7
6–12	62	16*	16	3	27	58.7
0–12	92	25*	19	3	45	67.2

* Subtracted from the general figures in computing Mortality.

SUMMARY

A study was made of the fate of one hundred and thirty infants under two years of age with pulmonary tuberculosis. Sixty-four of these died and sixty-six were living at the time of the last note on the history. Of these sixty-six, only thirty are known to have lived for a period of more than one year after the diagnosis was made. Some of these, however, were observed for periods of four and five years.

In computing the mortality, only those babies were classed as "Living" who were known to have lived at least one year after the diagnosis was made. This eliminated thirty-six babies and the mortality among the remaining ninety-four was as follows: Under one year of age, the mortality was 78.7 per cent; from one to two years of age, the mortality was 57.4 per cent; and for the two years, the mortality was 68 per cent.

As the fate of those babies who were observed less than one year is still uncertain, they are best classified in a distinct group.

The fate of the one hundred and thirty babies is shown in the following table:

AGE	DIED		LIVING		FATE UNKNOWN		TOTAL
	Number	Percent	Number	Percent	Number	Percent	
months							
0–12	37	60	10	16	15	24	62
12–24	27	40	20	29	21	31	68
Total.....	64	49	30	23	36	28	130

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A CASE OF RECURRENT PNEUMOTHORAX

M. I. MARSHAK AND JOS. W. CRAIGHEAD

From the Sanatorium of the Jewish Consumptives' Relief Society, Edgewater, Colorado

Although Hippocratic succussion is a sign which has been recognized as far back as medical history goes, the significance of the sign was at first misunderstood, as it was attributed to fluid per se. Spontaneous pneumothorax was placed on a proper basis in 1803 by Itard (1), who reported five cases of pneumothorax following pulmonary tuberculosis recognized at autopsy. Before his time, the reports show that most of the pneumothoraces recognized were those of the traumatic type. One interesting case of this period was that of Boerhaave's (2), which at autopsy showed pneumothorax following oesophageal rupture produced by vomiting. Riolan (3), Combalusier (4), and Pouteau (5) reported discovering pneumothorax, having heard the escape of gas while operating on empyema.

Laennec (6) in 1819, gave the first full description of the physical signs and symptoms present in spontaneous pneumothorax. Very little has been added to these signs since his time, although percussion has received a more prominent place in the diagnosis. It is claimed that he was the first to diagnose this condition in the living.

Many cases of spontaneous pneumothorax were reported in the succeeding years and a large amount of experimental work was done to prove the various causes of pneumothorax and to demonstrate the factors which produce succussion and the metallic tinkle. The gas in spontaneous pneumothorax was also made the subject of extensive experimentation, both as to production and composition.[1]

Recurrent pneumothorax seems to have been unrecognized until near the end of the nineteenth century. The first report in indexed literature of this condition was made by Austin Flint (7), in 1886, of a case which had two attacks with recovery. Hall (8), in an article on pneumothorax in apparently healthy persons, published in 1887, mentioned one case. Gabb (9), in 1886, reported an extremely interesting

[1] The early references are freely quoted from C. P. Emerson's article on Pneumothorax, Johns Hopkins Hospital Reports, Vol. XI, 1903, 1–450.

case in a woman fifty-six years old, who had three recurrences at intervals of six, two and five years. After this year reports although still few become somewhat more frequent. In 1889 West (10) reported a case with two and probably three attacks at seven and three month intervals. Knight (11) in 1890, described a case in a boy of sixteen who had had a previous attack twenty months before. In 1894 Vitvitski (12) gave an autopsy report which showed a pneumothorax existing, as well as a scar on the pleura where an attack had taken place one year previously. Goodhard (13), in 1896, reported a most unusual case which had had two attacks, the first on the right side, followed one year later by an attack on the left. In 1898 Finny (14) described a case with two attacks on the same side, fifty-eight days apart. In 1900 Vieryuzhski (15) described a case which had had a spontaneous rupture on the right side in 1894, clearing up in five weeks, but recurring a few weeks later and again recurring in 1899 on the left side. Rendu (16), while discussing a paper written by Variot and Roy in 1901, called attention to a case that he had which cleared up after fluid developed and recurred when fluid was aspirated. This was probably not a true recurrence, as the fluid no doubt acted as a trap and closed the opening, which when the fluid trap was removed again became patent. Both West (17) and Jochmann (18) reported cases in 1902. West's case had the first attack on the left side and the second, six months later, on the right. The same year Maillart and Lassere (19) reviewed the literature on spontaneous pneumothorax and collected seven cases of recurrence out of a total of fifty-one. Weber (20), in discussing the clinical forms of pneumothorax in 1903, mentioned a case of recurrent pneumothorax seen by his father, Sir Hermann Weber. Grenier (21) described a most interesting case in 1905 in a patient who had an attack of left-sided spontaneous pneumothorax in 1903, recurring on the right in 1904, then again in 1905 on the left side. In 1907 Sale (22) reported an extremely interesting case with a history of eleven attacks, he, however, having seen only one of these. Hamilton (23), in 1908, described a case which was perfectly well ten years after the last attack. In 1909 Jamieson (24) reported one case with two attacks. Bach (25) described one case in 1910. Nikolsky (26) in an inaugural dissertation (1912) collected sixty-eight cases of spontaneous pneumothorax, of which fourteen had recurrences. Hamman (27) and Sklodowski (28) both reported individual cases in 1914, Hamman's case having the first attack on the right side and a recurrence one year

later on the left. Pottenger, in discussing Hamman's paper, mentioned a case that he had seen with recurrence on the same side five years after the first attack. In 1915 Cummer (29) reported a case with recurrence on the same side after a five month interval. Hamman (30) reviewing the literature on spontaneous pneumothorax in 1916, gave the further course of the case he reported in 1914, which proved to be tuberculosis.

It is interesting to note here that since the Roentgen ray has become a factor in diagnosing lung conditions, more cases have been proved and reported than before the X-ray era.

Our case had three recurrences, proved by X-ray plates and by manometric readings.

Case report

J. B., admitted March 3, 1915. A far advanced case of pulmonary tuberculosis, Turban III (R_3 L_3), complicated by tuberculous larynx and copious haemoptysis. On November 6, 1915, an X-ray plate, taken for diagnosis because of severe dyspnoea, showed a pneumothorax from the left sternoclavicular articulation obliquely outward and downward to the axilla at the level of the fourth rib. The patient improved and felt fairly well until February 6, 1916, when she developed a sudden sharp pain on the left side with marked dyspnoea and cyanosis, the temperature remaining about the same as before the attack. An X-ray plate taken February 12, 1916 showed a pneumothorax extending this time obliquely downward from the junction of the inner and middle thirds of the clavicle to the axilla at the level of the sixth rib. A plate taken February 29 showed some decrease in the size of the pneumothorax, the pocket now extending obliquely downward from the middle of the clavicle to the fourth rib. Examination, May 12, 1916, showed no signs of pneumothorax. On August 13, 1916, the patient again developed a sudden sharp pain in the left axilla with marked dyspnoea, cyanosis and symptoms of shock, with sharp drop of temperature from 102° F. to 97° F., rising on August 16 to 100° F. Examination on August 13 showed signs of pneumothorax. A needle inserted on August 14 gave a manometric reading of -60 to $+20$ mm. of water. We withdrew gas until the reading was -70 to 0 mm. August 15 the reading was -30 to $+40$ mm. We again withdrew gas until the manometer showed -110 to $+5$ mm. On August 16 the patient was extremely dyspnoeic and in great shock, the manometric reading being -70 to $+80$ mm. and gas was withdrawn to a reading of -80 to $+5$ mm. On August 17 the morning reading was -100 to $+40$ mm. and we again withdrew gas to a reading of -100 to -30 mm. The afternoon of the same day we had to go in again and found a reading of -70 to $+30$, and withdrew gas to a

reading of −120 to −10 mm. after which she felt somewhat more comfortable. An X-ray plate taken August 25 showed complete reëxpansion. The patient felt fairly well, although bedridden, from that date until October 21, when she had another attack of pain, this time in the lower half of the left axilla, with dyspnoea and cyanosis, with no effect on the temperature. A plate taken October 31 showed a pneumothorax pocket extending from the eighth to the tenth ribs posteriorly and from a line two inches external to the mid-line internally to the ribs externally, with no evidence of pneumothorax above. There were no more attacks between October 21, 1916, and the date of death, November 26, 1916. No autopsy was obtained.

It is interesting to note that the greatest number of these recurrences took place in patients who showed no demonstrable tuberculosis and that the majority of them recovered. Those of Goodhard, West, Vieryuzhski, Grenier, and Hamman all had recurrences on the side opposite to that of the original spontaneous pneumothorax. The total number of cases here, including our own, is forty-three. However, there is some likelihood that Nikolsky's and Maillart and Lassere's collections might have overlapped, which would of course diminish the total number. Of the cases collected, those of Gabb, Sale, Vieryuzhski, Grenier, and probably one of West's, had more than one recurrence.

We should like to call attention to the fact that we treat all our spontaneous pneumothorax cases by withdrawal of air until the patient is comfortable and usually produce a negative manometric reading. Later, if the condition of the opposite lung warrants, and if the collapsed lung showed evidence of tuberculosis before the spontaneous rupture occurred we continue the pneumothorax.

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EDITORIAL

COMMUNITY STUDIES OF TUBERCULOSIS

In discussing any phase of civilization that may have an effect on community health it is our common and unfortunate habit to deal too much in asseveration and too little in what might be called a healthy appreciation of the great complexity of the particular problem. Many of us are perfectly competent to interpret what we read on the chemical balance or to draw proper conclusions from observations made on guinea pigs in the laboratory cage. But in approaching any situation that concerns large numbers of people who live under conditions of highly organized life we seem to forget the enormous number of heterogeneous forces that undoubtedly contribute to the moulding of a common result. We are all too prone to try to hew a short and direct course through what is really a fog of ideas. If enough of us agree, we think that we have found Truth. Only too often, however, a new discordant fact or two clears the atmosphere and shows us that in our long and comfortable journey we have proceeded very little beyond our starting point.

There is hardly any doubt that any habit of thought in an individual is the resultant of countless determinants not only in his own life history but also in those of his ancestors. If this is true, how numerous and perhaps divergent must be the factors that in many instances finally produce definite and permanent alteration of tissue and function, and thus symptomatic disease. It is almost a medical aphorism that a certain type of cirrhosis of the liver is due to the ingestion of alcohol. Yet how few alcoholics develop this cirrhosis! It is also undoubted that some men will not have gout if they eschew beer. But how few beer drinkers ever get gout! What determines the chronic nephritis, the angina pectoris, the cancer, in certain individuals? We say that certain irritants produce them. True; but many other factors must be at work, factors that are perhaps more decisive than the irritant. Relatively many more parous women are stricken with cancer of the breast and cervix uteri than are virgins. If we consider only this feature the cause, the irritant, is perhaps plain. Yet the fact remains that not infrequently virgins and non-parous women do acquire breast and cervical cancers. Or let

us take simpler instances. What determines localizations of the same irritant? Why tabes dorsalis or gumma of the liver or aneurysm or aortic insufficiency or tibial periostitis? The inference is almost compelling that in so many cases once having discovered a known and external cause of disease and tissue change we have not even half appreciated its total cause. We have perhaps only begun to approach its real cause. In individuals, in families, in races there are characteristics, both acquired and inbred, that finally decide just where and how man's external enemies shall localize and develop.

In the study of any disease too much stress cannot be laid on the study of man himself. It is therefore particularly encouraging that during the last few years we, in tuberculosis, have been breaking our bonds and have more and more shown the tendency not to remain satisfied with the mere discovery of the tubercle bacillus in any particular instance. A house or a city block swarming with tubercle bacilli may or may not mean a great deal. But it will not explain all that there is to all cases of tuberculosis in infants, adolescents and the aged that come out of that house and city block. So much we are sure of. And we sometimes wonder whether in and of itself it will explain very much more than the opportunity for infection.

That we are thus breaking our bonds is becoming increasingly plain. For the time being we are supplementing the test tube and the experimental rabbit in something more than a speculative way. Community studies are becoming fashionable. We are now reading of the Michigan Survey, the Framingham Experiment and the Saranac Lake Census. All of these, if done properly, will take money. It is the writer's opinion that the importance of the studies will justify the expenditure of any conceivable amount of money; that is, if the investigators approach their problems free from all bias and prejudice, if they appreciate its difficulties in even the smallest and most provincial communities and if their enthusiasm runs more to hard work and capable analysis and less to dogmatic conclusion and roseate anticipation of immediate results. We cannot have too much information. We cannot have too little premature classification and systemization.

The Framingham Experiment is already well under way. At the last meeting of the Board of Directors of the National Association, Dr. Armstrong, the executive officer, reported that all elements of the population of the city were lending their earnest coöperation. Not only is there a large local advisory committee at work on the completion of

plans for the methods of attack, but the assistance of the State Health Department, the United States Public Health Service and neighboring institutions has already been enlisted. Local insurance agents and the school physician and nurses have finished a sanitary survey and have entered upon a census of the sick; and it is their intention to have all persons whose health is below par undergo physical examination. These examinations will be made under the auspices of the local medical club which includes all the physicians, who for doubtful cases or tuberculous suspects will have the assistance of consulting specialists. An educational propaganda carried on through many channels is preparing the ground for effective work and making ready the people for the enactment of future measures. There are committees on diagnostic standards, health week and prize essays which lend assistance to the director while the medical club is planning a campaign of instruction which will comprise many lectures and clinics on tuberculosis.

It can easily be seen that besides any new information concerning the etiology and prevention of tuberculosis that would be gained by such measures, the experiment is certain to bring about a definite and lasting awakening among the people as to the real nature and meaning of tuberculosis as a community problem.

The Saranac Lake study is of a different nature. The acting commissioner of the Department of Health of New York State has requested the boards of health of those municipalities that are resorted to by tuberculous patients to undertake a health census of their respective communities for the following purposes: (1) To discover and locate all unreported cases of tuberculosis. (2) To ascertain the present condition of health of all tuberculous patients together with their housing, working and social conditions. (3) Because of the greater liability of infection in childhood, to obtain a complete record of the number, ages and conditions of health of all children in these families and ascertain the degree of exposure to the disease under which these children live.

The Saranac Lake Board of Health has accordingly begun such a survey. It plans nothing more nor less than a complete medical history of every resident, transient and permanent, of the village.

The result of this census should be of interest for several reasons. As a resort for the tuberculous Saranac Lake has now an uninterrupted history of about thirty years. For its size it is perhaps as pure a type of a tuberculized community as this country affords. It really began as a health resort and its growth and development have been due almost

wholly to the tuberculous and their families who have settled there. It has no manufactories and there has been hardly any influx of population except by those in search of health. There are now numerous young adults who were born in Saranac Lake of tuberculous parentage and who grew up with the disease around them on every hand. In a way the village offers as pretty an opportunity for the study of a definite phase of tuberculous infection and its results as one would wish. It would be particularly valuable if the authorities could also trace those who after early life in Saranac Lake have moved away. It would be extremely interesting to discover how such people have withstood the stresses of more complex city life.

It is to be hoped that studies such as these may teach us much. They should be a model and an example to other enlightened communities. It is hardly to be doubted that wherever tuberculosis is studied in an intelligent way the people whose aid counts will lend every assistance in their power.

A. K. K.

MEDICAL NOTES, ABSTRACTS AND REVIEWS

It will be the eventual purpose of the REVIEW thoroughly to canvass all literature on tuberculosis as it appears in other medical periodicals and to present it in abstract form. Authors are requested to send abstracts or reprints of their papers to the Abstract Editor, Dr. George Mannheimer, 41 West 51st Street, New York, N. Y. In the present number many of the abstracts have been taken from other medical publications, such as the Journal of the American Medical Association, the Medical Record, the New York Medical Journal, etc., to whom acknowledgment is due.

Treatment of Tuberculosis in Infancy.—The primary focus or entrance point of tuberculous infection cannot be demonstrated during life; the primary lesion is represented therefore by a localized mass of lymph glands which can be seen and felt in exposed places like the neck, axilla, groin or abdomen; or made out in the chest by auscultation, percussion and the x-ray. The Pirquet test is corroborative evidence; but it may be necessary to make a subcutaneous test.

There are three factors essential in the treatment, rest, fresh air and sunlight, suitable food. Rest should be persisted in till long after the symptoms have been relieved, and then abandoned gradually. The regions drained by these primary glands should be protected from all diseases in order to avoid the waking up of the tuberculous process and its spread. The treatment of tuberculosis in infancy consists essentially of the treatment of the primary lesion and the prevention of its spread. As long as this is successful the infant is safe; and unless tuberculosis is recognized earlier than the secondary forms, little or nothing can be done in treatment.—*Treatment of Tuberculosis in Infancy, W. H. Howell, Boston Med. and Surg. Jour., September 21, 1916.*

The Von Pirquet Reaction in the Schools of Lausanne.—Weith, after a general outline of the results of this reaction and of the incidence of tuberculosis, states that Lausanne has a comparatively low death rate for this affection, which is virtually stationary. In comparison with other Swiss cities, however, it is high, and it has been proposed to use the von Pirquet in the schools. Is it wise to test thousands of children with the result that a few more lives may be saved? According to authorities 95 per cent of all children are tuberculous, *i.e.*, have the condition in a latent form. What would be the good if all were "pirquetized?" By rights, according to theoretic conditions, one would need to be "pirquetized" weekly, for those not infected could readily become so. Would a negative reaction guarantee that a child was not tuberculous? The advocates of the test admit that such children as are candidates for tuberculosis betray this lack of resistance in many ways. These are the real suspects. Nevertheless should universal tests be practised? To cause a reduction in the incidence of tuberculosis prophylaxis seems the logical course. This means general sanitation, rigidly carried out, plus school hygiene. The author and other opponents of "pirquetization" are not opposed to the test *per se, i.e.*, they have no fear of complications, nor do they shrink from the physical effort, nor are they old fogies. The task, however, increases with each year because both positives and negatives must be followed up, along with all new pupils. There can be no common ground between "pirquetization" and vaccination for smallpox, for the latter is meant to protect directly the individual and the public.—*A propos du Pirquet dans les écoles de Lausanne, Weith, Rev. Med. de la Suisse Rom., No. 9, September 20, 1916.*

Chronic Influenza in Pulmonary Tuberculosis.—Hamblet and Barnes endeavored to obtain data as to the frequency of influenza infection as a complication of phthisis; as to the frequency with which the symptoms of patients diagnosed as having pulmonary tuberculosis, but with negative sputum, may be accounted for by chronic influenza infection, and as to what extent the percentage of recoveries in sanatoriums for tuberculosis have been swelled by the inclusion of cases of influenzal bronchitis. Of 100 patients, diagnosed as having closed tuberculosis whose sputums were cultured for influenza, at least 96 were negative. Of 100 patients, diagnosed as

having closed tuberculosis, influenza bacilli were recovered from six. Of 100 patients, with open tuberculosis, influenza bacilli were recovered from seven. Of 20 patients in whom the diagnosis of tuberculosis continued doubtful, the sputum was negative for influenza bacilli in all. In none of the patients examined did the influenza bacilli occur in pure culture nor were they the predominating organism.—*Hamblet and Barnes (Waltlum Lake, R. I.), Arch. of Int. Med., September, 1916.*

Case of Pulmonary "Calculi."—Description of a case of this clinically rarest of affections, which is termed in German *Steinhusten* because stones are coughed up by the victim. Autopsy finds are not included, for these are not so infrequent; but of clinical cases few have ever been placed on record. In the most recent, reported by Bickel and Grunmach, the stones were no larger than a pea. The author's patient was a woman of 58, healthy until recent years, when she began to suffer with symptoms suggesting influenza. During the first attack no "stones" were coughed up, but during a second visitation she began to expel small "stones." She has never since been entirely free from cough. A third attack of "influenza" one year after the preceding led to paroxysms of coughing, with dyspnea, and finally in expulsion of what she termed a "bone." About 8 months later a fourth attack culminated in a like manner, while at the end of another two weeks a fifth seizure brought the patient for the first time under medical care. She brought with her four of the "stones," two of which were as large as hazel nuts. An x-ray showed two circumscribed shadows in the lung which doubtless represented unexpelled "stones. Microscopic section showed the presence of bone corpuscles and lamellae in all the foreign bodies. This surprising find really furnished an explanation of the whole disease process which must have been tuberculous. The patient had developed latent calcified foci of the disease, which in turn had led by processes known to pathologists to a metaplasia of osseous tissue as a result of constant irritation of the fibrous capsule of the focus. The "stones" expelled were in reality osseous tissue. While a "stone cough" may be a reality, the present case should be termed "bone cough."—*Ein Fali von Steinhusten, Marie Helbig, Münch. Med. Wochenschr, October 17, 1916.*

Coryza in Early Tuberculosis.—During the early invasive or latent tuberculous stage one encounters a peculiar coryza which, it appears, is caused by excretion of toxins from the circulation; the toxins, while having a predilection for the nasal mucous membrane, cause a reaction of the mucosa. A sustained coryza should make one think of a possible tuberculous nidus. In a minor degree there is some resemblance to hay-fever, but the lacrymation is not present. The tuberculous focus will often be discovered within the area encompassed by a circle whose radius extends three inches from the thyroid notch. Waldeyer's ring is a fertile field for tuberculous processes.—*Rhinal Premonstration of Tuberculosis, J. A. Hagemann, Med. Record, December 30, 1916.*

Tuberculosis of Bronchial Glands.—The symptoms are caused by the absorption of toxins and by pressure on adjacent structures. The syndrome of tuberculous toxemia is the same as that from the lungs; it is the pressure symptoms that are of particular interest in this form of tuberculosis. Compression of the great vessels may cause cyanosis and edema of the face or even edema of, and hemorrhages into the meninges. It is possible that compression of the superior vena cava may have something to do with the rapidly fatal cases of cyanosis, dyspnea, unconsciousness and death, so typically fatal in the bronchial gland tuberculosis of infants. It is also possible that the stasis in the meningal circulation explains the association of tuberculous meningitis with that of the bronchial glands. As a rule, the meningitis attacks children in apparently good health, and without the knowledge acquired at autopsy there would be no reason for suspecting that meningitis may be secondary to absolutely concealed tuberculous processes in the bronchial glands. Autopsies teach that this association always exists, and it is inferred that the rapidity of meningeal processes leaves no time for slower developments. Pressure upon the sympathetic may cause irritability of the vesamotors, evidenced by rapid changes from pallor to blushes; unequal or unilateral flushing of the cheeks; circumscribed reddening of the second intercostal space, associated with menstrual rises in temperature, a consciousness of unstable heart action, of flutterings and palpitations and quickened pulse; unequal pupils and protrusion of the eyeballs. It is probable that a certain class of gastrointestinal disturbances, characterized by lack or suppression of functional activities, may be due to pressure upon the sympathetic nerves, because these disturbances are present in tuberculosis of the bronchial glands when pulmonary tuberculosis cannot be proved. It is possible that the rhachialgias and intercostal neuralgias

preceding the discovery of tuberculosis of the spine may also be caused by enlarged bronchial glands and the infection of the spine may prove to be a secondary condition. Pain in the region of the trapezius is one of the earliest symptoms of tuberculosis of the bronchial glands, as is also pain over the root of the lung. Pressure upon the fibers of the recurrent laryngeal may cause pain in the larynx, laryngospasm, and paralysis of the vocal cord. Changes in the quality of the voice may be one of the very early manifestations of enlarged bronchial lymph-nodes.—*Tuberculosis of the Bronchial Glands, Mary E. Lapham, N. Y. Med. Jour., October 21, 1916.*

Radiography of the Bronchial Lymph Nodes.—The bronchial lymph nodes, anatomically, are divided into four groups: (1) Tracheobronchial; (2) bifurcation; (3) hilum, and (4) pulmonary. Clinically, cases are grouped into four divisions: (1) Normal, with no physical signs; (2) with right paravertebral dullness; (3) with double paravertebral dullness; (4) showing parasternal dullness on one or both sides. In the series of cases examined radiologically, every case was examined in three positions: dorsoventrally, ventrodorsally, and right semilaterally. The method of diagnosing the different forms of nodes is given. As the result of the examination of numerous individuals the following deductions were made: (1) That thoracic tubercle is widely distributed in children of school age in thickly populated urban districts; (2) there are notable powers of resistance which can control these processes even after extensive dissemination throughout the lung; (3) that serious chances of recrudescence exist under conditions of stress which these children must combat in adolescence and adult life; and (4) there is mutual interdependence of clinical and radiological methods of examination in complete investigation of the tuberculous thorax.—*Radiography of the Bronchial Lymph Nodes, Walker Overend and Clive Riviere, Arch. of Radiol. and Electrother., August, 1916.*

Relationship of Tuberculosis to Psoriasis.—Gaucher adduces evidence to show that the initial cause of psoriasis is tuberculosis, or, more precisely, tuberculous toxic infection. From the pathological standpoint a bond between the two conditions is suggested by the cases of psoriasiform papules to which the term parapsoriasis has been applied and some of which have been thought due to a papular skin tuberculosis. The pathogenesis of psoriasis may be compared to that of lupus erythematosquamosus, many cases of which are unquestionably of tuberculous causation. The chief argument in favor of the tuberculous origin of psoriasis is, however, derived from clinical observation. G. has been struck by the frequency of psoriasis in tuberculous families. The children of a single family often present psoriasis and tuberculosis in different individuals. Psoriatic patients not infrequently develop tuberculous lesions. In all cases of acute or scarlatinoid psoriasis which had become transformed into secondary pityriasis rubra G. witnessed ultimate death from pulmonary tuberculosis. Many psoriatic patients have lost a parent, brother, or sister from the latter disease or from tuberculous meningitis, or have relatives with osseous or articular tuberculosis. The children of parents with psoriasis not rarely succumb to tuberculous meningitis or later to pulmonary tuberculosis, or exhibit lymphatic, bony, or articular tuberculous disease. Psoriasis patients themselves sometimes develop pulmonary tuberculosis, or, more often, show signs of larval tuberculosis, such as chronic cervical adenitis, asthmatic states, and chronic joint disorders. Similar evidence was, for a long time, available to show the tuberculous nature of lupus, now recognized as tuberculosis by all. The presence of tubercle bacilli or of the tuberculous follicle is no longer essential in making the pathological diagnosis of a tuberculous lesion. In fact, atypical, inflammatory, or nonfollicular tuberculosis appears to occur as frequently as the typical form.—*Les rapports du psoriasis avec la tuberculose, M. Gaucher, Bull. de l'Acad. de Med., No. 44, November 7, 1916.*

Intestinal Stenosis of Tuberculous Origin.—The four patients were men of 35, 44 and 54 and a woman of 50. The stretch of bowel involved was resected in three of the cases. In the older man the small intestine showed numerous ringlike strictures at intervals of about 30 cm. He had been having colic pains with distention and exaggerated peristalsis for nearly three years, with vomiting. The exploratory laparotomy revealed the serosa smooth but with nodules near the strictures. The latter were of half solid (*halvfast*) consistency. The mesenteric glands seemed normal. The abdomen was closed without further intervention, and great improvement followed at once, but the later history is not known. The single stricture in the woman was resected. It was free and movable, like a cicatricial stricture, and for eleven years after resection of this stretch of the ileum she was in good health, free from pains. Before this she had suffered from

digestive disturbances for twenty-two years, with colic pains, tympanites and exaggerated peristalsis with vomiting. Of the two other patients one had had abdominal pains recurring during eight years, the symptoms gradually presenting König's complete syndrome of tuberculous stenosis. Two tumor-like strictures were found in the lower ileum. Death occurred a year later from tuberculous peritonitis. In the fourth case there had been symptoms for only three or four months, and they were merely irregularity in bowel function, difficulty in passing flatus, and eructations. A tuberculous tumor was found in the cecum and a stricture near the valve of Bauhin. Death followed on the ninth day from peritonitis, one of the sutures having given way. The fact must never be forgotten that tuberculous strictures are very likely to be multiple. The only chance for recovery is from an operation.—*Tuberculous Stricture of the Small Intestine; Four Cases. (Till de tuberkulösa tunntarmsstrikturernas kasuistik.) P. Carlsson, Hygica, LXXVIII, No. 19, 1916.*

Tuberculosis of Mammary Gland.—Report of four cases of mammary gland tuberculosis. Since the classical description of its microscopic pathology by Dubar in 1881, about 180 undoubted cases have been reported, but little has been added to the known pathology. Dubar described two types under the terms nodular or discrete, and confluent. The nodular type has been subdivided again into the solitary and disseminated forms, but these are often but different stages in the same case. The disseminated nodules may become confluent, with the formation of large caseating areas. There may be a single nodule of firm consistency giving the impression of an adeno fibroma. In the discrete variety, as a rule, the breast does not increase in size, and the skin is unbroken. The nodules consist of zones of blue or bluish gray tissue surrounding yellowish or wax colored centers. If multiple, the foci are scattered in normal gland tissue. Scott has described a type in which the chief feature is a sclerosis. This usually occurs in old women and the microscope is necessary to definitely diagnose it from cancer. It may be compared to fibroid phthisis with the absence of cavities. Most cases of breast tuberculosis have broken down, showing fluctuation, or have gone further to abscess formation with multiple sinuses. This is the confluent variety, and the breast is usually symmetrically enlarged. Retraction of the nipple is nearly always present, occurring early in the disease, and the lymph nodes on the corresponding sides are usually enlarged. About 60 per cent of cases have been reported as primary. This appears to the author to be a misnomer, and he believes that they are undoubtedly secondary to a primary focus elsewhere in the body, and occur either from direct extension, hematogenous invasion, or retrograde lymphatic processes. There may be a few cases in which it is primary, such as one reported by Deaver in which a nurse fell on an infected instrument. There is no record, however, of a patient coming to necropsy with tuberculosis arising in the mammary gland and confined to the breast. Halstead and LeCount believe that the progress is retrogressive, from the axillary lymph nodes in a large percentage of cases, but the majority of observers seem to be inclined to consider the infection through the blood. The gross picture of the confluent type is rarely confused with anything except actinomycosis or tertiary syphilis. Microscopically the process resembles tuberculosis of other similar tissues. Some fibrosis occurs in almost every case, but it is much more marked in that form described by Scott in which no abscess formation occurs. In this variety the presence of bacilli is most difficult to demonstrate, though there is difficulty of finding bacilli in the tissues generally in tuberculous mastitis.—*Tuberculosis of the Mammary Gland, Gatewood (Chicago), Jour. A. M. A., December 2, 1916.*

Tuberculous Meningitis Simulating Multiple Sclerosis.—Cammarata relates the following case: The patient was a boy aged 3 years, of good family and personal antecedents. Six months before consultation he exhibited a tremor of the right hand, worse in the daytime. Three months later his right leg lost its feeling and he walked with a tremulous, spastic gait. On examination he showed an intention tremor and could not raise a glass to his lips unless aided by the left hand. Meningitis could be excluded completely. Diagnosis, sclerosis *en plaques*. Some weeks later he was attacked by fever, headache, and vomiting, stiff neck, convergent strabismus, terminating after a few days in convulsions. Lumbar puncture was now practised. The fluid emerged under strong pressure and the sediment contained tubercle bacilli. There was some temporary improvement, but death followed an exacerbation. Autopsy was refused. The connection between the early symptoms of sclerosis and the meningitis was obscure. Sachs describes an abortive type of the former, which, however, is extremely rare at so early an age. By exclusion it is quite impossible to reach any other diagnosis. A possible compromise

diagnosis would be a focus of tuberculous meningitis behaving like a patch of sclerosis.—*Meningite tuberculare simulante nel suo periodo prodromico la sclerosi a plache, A. Cammarata, La Riforma Medica, October 30, 1916.*

Ocular Tuberculosis.—Of ten cases comprising scleritis, sclerosing keratitis and sclerochoroiditis, tuberculosis was definitely present in eight. This was shown by a reaction following tuberculin inoculations in five, all the patients who were inoculated reacting; in three there were signs of pulmonary tuberculosis; in one case signs of an old pulmonary process, and in one case the examination could not be completed. All four patients with choroiditis reacted to tuberculin injections. Each of the ten cases, four of scleritis, two of sclerochoroiditis and four of choroiditis, in which the blood Wassermann test was performed, reacted negatively, while the nine of these patients inoculated with diagnostic doses of tuberculin, reacted positively. In one patient not inoculated, the physical signs of pulmonary tuberculosis were prominent. The seven patients in whom a general reaction followed the diagnostic use of tuberculin, showed negative physical findings. The eighth patient has yet to be roentgenographed. Of the nine reactions to tuberculin, eight were general, without an ocular focal reaction, while one was an ocular focal reaction without a general reaction. All six of the patients in whom the positive Pirquet reaction was followed by a diagnostic inoculation of tuberculin, reacted positively to the latter. Tuberculin therapy in the five cases in which it was employed, accomplished the apparent cure or marked improvement not attending the use of the routine measures.—*Ocular Tuberculosis; Report of Cases, R. B. Metz (Cleveland), Cleveland Med. Jour., September, 1916.*

Diagnostic Value of Tubercle of the Choroid.—There is a tendency in modern times to overlook the general diagnostic value of tubercle of the choroid. In three recent cases an acute tuberculosis was diagnosed as such by the discovery of tubercle of the choroid. In one the diagnosis was made from an apical pneumonia; in another, from a pneumococcal peritonitis, and in a third, from typhoid fever. The finding of these three cases within so short a time lends truth to the statement that acute tubercle of the choroid is by no means an uncommon manifestation if looked for in the right places and in the right way. A more frequent ophthalmoscopic examination of children in hospitals would prove a great aid in diagnosis.—*The Diagnostic Value of Tubercle of the Choroid, Sidney Stevenson, Lancet, September 9, 1916.*

Diagnosis of Bone and Joint Tuberculosis.—Forty-nine fine radiograms accompany this article, but Forssell emphasizes that negative Roentgen findings by no means exclude tuberculosis. The chief advantage of Roentgen examination is in cases which are apparently tuberculous but in which some other origin is shown by the rays. A number of instances of this are described, and the special points for differentiation are analyzed as the roentgenograms are compared especially those showing Köhler's and Schlatter's diseases, dissecting osteochondritis, chronic traumatic arthritis and syphilitic lesions. Future progress in this line will be by minuter persevering study of the roentgenograms in connection with the pathologic anatomic findings and by taking the pictures from more standpoints. Many of the supposed "failures" in Roentgen examination come from our inability to see, and to interpret correctly what we do see.—*Några ord om benoch ledgangs tuberkulosens diagnostik, G. Forssell, Svenska Lä Karesällsk Kapels Handlingar, XLII, Nos. 4, 1916.*

Fatal Hemorrhage in Bone Tuberculosis.—Report a case of tuberculous involvement of the lungs and many bones and joints and glands in various parts of the body. Uncontrollable hemorrhage developed from a draining sinus in the sternum. After a post mortem study it was thought that the tuberculous process had extended into the internal mammary vessels.—*R. C. Patterson, Amer. Jour. of Orthopedic Surg., October, 1916.*

Lupus Erythematosus and Tuberculosis.—The opinions of leading dermatologists in 1907 according to Civatte could be classed into three groups: Those who held the disease to be of tuberculous origin; those who held the contrary view; and those who thought that in some cases the disease is of tuberculous origin and in others not. There were 17 members in the first group, 25 in the second and 7 in the third. Gougerot, Ehrman and Reines succeeded in producing tuberculous lesions in guinea pigs by the inoculation of tissue removed from lupus erythematosus lesion. Bloch and Fuchs deserve the greatest credit for their epoch making experiments. They obtained a corpuscle-free extract of the diseased tissue, and by placing this into the skin of individuals who reacted to tuberculin, obtained in every case a reaction both by the cutaneous and intracutaneous

method. The papules resulting from the latter inoculation were examined histologically and showed the tubercle structure. They also placed pieces of tissue from the skin lesion into the peritoneal cavities of guinea pigs with resulting positive reactions in some of the cases. They believe that the disease, of chronic type, is due to an infection with the tubercle bacillus, perhaps of lower virulence or of a different variety, upon a soil which in the majority of cases reacts in an atypical manner.—*Lupus Erythematosus and Tuberculosis; a survey of the literature. L. B. Mount, Med. Record, December 2, 1916.*

Tuberculosis of the Tongue.—A soldier, 32 years old, whose one sister and one brother had died of tuberculosis, had frequent short attacks of bronchitis and diphtheria in childhood; was a moderate drinker and cigarette smoker. Four years ago he noticed a small elevated white area on the left border of the tongue, and was advised by a surgeon to apply tincture of iodine twice a week. Physical signs in both lungs. Patient well nourished, muscular; movements of chest equal on both sides; posterior cervical, epitrochlear and inguinal lymph nodes enlarged; temperature, pulse and respiration normal. The tongue was slightly enlarged, elevated, corrugated and ulcerated; the ulcer occupied the posterior two-thirds, not painful. The sputum contained tubercle bacilli. Microscopically, the sectioned specimen showed absence of epithelium over the ulcerated area, beneath which was an accumulation of young fibroblastic cells surrounding a small area of necrosis. There were scattered foreign-body giant cells surrounded by accumulations of round cells. Tubercles of various sizes were found in the connective tissue around the muscle bundles and in the muscle proper. Numerous acid-fast bacilli were present, some in the giant cells. Diagnosis: Tuberculosis of the tongue.

In a series of 231 cases collected from the literature, only 26 were undoubted cases of the primary form of the disease. The condition is very indolent. The ulcerated area does not enlarge with any degree of rapidity, and does not respond to local treatment. Enlargement of the tongue and pain come on late in the disease. The pain finally becomes severe, especially on swallowing and phonation. The tongue is covered by a glary, grayish mucus, and there is salivation. The most common site is on the superior surface near the tip. The neighboring lymph nodes are frequently involved. In the differential diagnosis, we must exclude simple ulcers which respond to local treatment; syphilitic ulcers by the history, Wassermann, the presence of other lesions and the antiluetic treatment; and carcinoma. The latter is difficult to exclude except by a microscopic section. The disease is more common in males, especially in the fifth decade. It is most commonly secondary, and may assume different types besides the ulcerated form, such as the fissured, granulomatous and papillomatous form. The medical treatment should only be followed in advanced cases or where there is a generalized tuberculosis. Complete excision of the diseased and the surrounding healthy tissue for some distance constitutes the rational treatment.—*Tuberculosis of the Tongue, J. R. Scott, Amer. Jour. Med. Sciences, September, 1916.*

Renal Tuberculosis.—The slight irritability of the bladder which does not yield to ordinary therapeutic measures is just as suggestive of renal tuberculosis, as the slight cough is of pulmonary tuberculosis. The process in the early stages is generally unilateral; nephrectomy in the early cases gives good results. In 80 to 90 per cent of cases the initial symptoms are those of a very severe cystitis; the desire to urinate frequently must be satisfied at once or there may be an involuntary emptying of the bladder. There is polyuria, the urine is pale, of low specific gravity, acid and shows pus and albumin. In 25 per cent of cases there is hematuria. In 20 per cent there is pain in the affected kidney and there may be a tumor. If there is no pus in the urine, tuberculosis can fairly positively be ruled out. If present, bacilli should be searched for. These may not be easy to detect. The inoculation test is also not infallible and consumes a good deal of time. Catheterization of the ureters is the best test. This may lead to a diagnosis even where the ureters appear to be occluded from the picture of the bladder and ureteral orifices. Nodules of the prostate, vas or epididymis are also very strongly suggestive. Palpation of the thickened ureter by vagina or rectum is fairly conclusive evidence of the side affected; so is the finding of a kidney mass—due to enlargement or hydronephrosis, provided the presence of renal tuberculosis has been proven. Radiography may disclose the outline of the kidney and calcareous masses. Pyelography should only be used in cases where there is doubt. The subcutaneous tuberculin test may aid in that besides being positive, it may occasion pain in the affected kidney.—*Diagnosis of Renal Tuberculosis, Thomas F. Laurie, N. Y. State Jour. of Med., October, 1916.*

Renal Tuberculosis.—Thirty-three personally observed cases were studied. In 20 per cent there was a history of other tuberculous lesions, and in 15 per cent, of pulmonary involvement. Cervical adenitis was noted once, old healed bone tuberculosis three times and a discharging ear of fourteen years duration once. Cystitis was the first symptom in 50 per cent of cases and if hematuria is considered as a symptom of cystitis, this condition was the first sign in 85 per cent of cases. Macroscopic hematuria was the first symptom in 15 per cent, while 50 per cent had macroscopic hematuria at some stage of the disease. The other 50 per cent had red blood cells in the urine microscopically. The bleeding is vesical and marks the beginning of an acute cystitis or the exacerbation of a chronic cystitis. The hematuria is usually early, lasts one to four days and, if it recurs does so within a month. The chief complaint was cystitis in 65 per cent, renal or ureteral pain in 35 per cent of cases. Symptoms of cystitis were a source of trouble even where other symptoms predominated, and most often indicated nephrectomy. The operation should be performed before there is extensive bladder involvement. The renal pain is usually colicky similar to that of calculus. Pyuria was present in every case. Tubercle bacilli were found in slide preparations in 85 per cent of cases, though repeated examinations were necessary in some instances. A positive guinea pig reaction was never found where no bacilli could be demonstrated in the smear, and in two instances the smears were positive while animal inoculation was negative. In 15 per cent the temperature was slightly elevated and in the rest entirely normal. In only 15 per cent was enlargement of the affected kidney noticed, while in a few there was compensatory enlargement of the healthy organ. In all except very early cases there were characteristic tuberculous changes in the bladder. Before bladder involvement there may be great frequency of urination. In such cases the finding of bacilli and catheterization to determine from which side they come is necessary. The cytoscope shows quite early reddening and induration of the ureteral orifice which becomes irregular, the induration extending until it involves the trigonum on the affected side. About this time the other ureter appears on the anterior wall as an indurated area. Later the affected ureter becomes stiffened and shortened and that portion of the trigonum is retracted. Where ulceration takes place the bleeding is more marked and the bladder discomfort greater. Miliary tubercles have not been met with; multiple ulcers were found in 15 per cent of cases. The involved ureter, except in early cases, could be palpated by vagina in 45 per cent. The general health continued good in 50 per cent, fair in 35 per cent, and poor in only 15 per cent of cases. In the latter there was lung involvement or other complications. All except four were treated by nephrectomy. The method of dealing with the ureter had no influence upon the percentage of sinuses resulting, except that one case treated by excision of the ureter down to the bladder has proved very satisfactory and the technical difficulties have been small. There have been no operative deaths, and only two have died since operation, one of pulmonary and one of miliary tuberculosis. About 30 per cent have had post-operative sinuses for periods ranging from several weeks to a few months; one for two years with no apparent impairment of health. Thirty-five per cent have been cured and are in good health. The others have improved. The continued bladder involvement has kept these out of the cured group. It appears that patients with advanced tuberculosis and those who had the disease for a long time have done better than those who had the trouble for a short time. This may be due to insufficient immunity in the latter and to loss of useful kidney tissue. A brisk rise in temperature following operation in a number of cases was probably due to a tuberculin reaction following the removal of tuberculous tissue. Of all cases, 45 per cent occurred between the ages of twenty and thirty; and 65 per cent, between twenty and sixty years.—*Renal Tuberculosis, H. D. Furniss, N. Y. State Jour. of Med., November, 1916.*

The Complications of Renal Tuberculosis.—Tuberculous cystitis is the necessary consequence, the first complication of renal tuberculosis, and by itself alone reacts intensely on the patient's general health. Surgical interference, such as cystotomy, will have a tendency to improve the vesical lesions and by placing the organ at rest the severe and constant pain is done away with. The excellent results of this operation can hardly be overestimated. The tuberculous lesions of the ureter produce stricture resulting in stagnation of the urine above. The importance of these lesions in renal tuberculosis is great, yet they are often overlooked. A pyonephrosis thus set up may be purely tuberculous, or, on the other hand, be due to a secondary infection. In the latter case the clinical picture may very likely be that of an ordinary non-tuberculous pyonephrosis.

This dual infection may also take place in the perirenal fat and the presence of tuberculous lesions can be demonstrated, although it may appear healthy and play an important part in fistulae following nephrectomy. Koch's bacillus may produce true cold abscess in the perirenal fat and such collections must not be mistaken for abscesses having their starting point in the ribs or vertebrae. These abscesses are sometimes the first evidence of renal tuberculosis, but in most cases they are due to a secondary infection, particularly by the colon bacillus, the organism reaching the perirenal fat by way of the blood or lymphatics. The evolution of these perinephritic collections is most variable. As they usually extend downward toward the abdomen they may simulate an appendicitis or open into some hollow viscus or even through the abdominal wall, when the resulting fistula remains permanent.

The extension of the infection toward the thorax is less common, although cases have been met with, and the real origin of the pleural or pulmonary suppuration will be difficult to detect. Pleurisy during a renal tuberculosis is not uncommon, and a careful history of the case will often reveal this lesion in the patient's antecedents before the renal lesion has given rise to any symptoms. In this case the pleurisy will usually be on the same side as the affected renal gland. A bilateral infection of the kidneys, at least at the beginning of the renal process, is relatively rare and is a great handicap to surgical treatment. When the lesions are of mild degree in one kidney, however, it is well known that an operation on the severely diseased renal gland may produce a great improvement in the patient's health.—*Editorial, N. Y. Med. Jour., September 23, 1916.*

Genital Tuberculosis in Male.—Postmortem and clinical findings show that the great majority of cases of genital tuberculosis have active tuberculosis elsewhere in the body, the infection in the genito-urinary tract being secondary. The majority of cases of tuberculous epididymitis have tuberculosis of the vesicles and prostate on the corresponding side, whether the condition can be demonstrable by physical examination or not. Cases of genital tuberculosis often have associated tuberculosis of the bladder and kidney, and a cystoscopic examination with catheterization of the ureter should be a routine procedure, in each case, before the possibility of such associated infection can be eliminated. The best treatment for the local condition, in most instances, is to remove the scrotal focus by epididymectomy or castration, and this should be followed by injecting the vas with a drachm of crude phenol, with the hope of eradicating the disease from the genital tract. The destruction of the local focus by this procedure is but the first step in the process of immunizing the patient against fresh outbreaks of the disease. Hygiene and tuberculin should be made use of indefinitely, as they serve further to aid, in a rational way, the immunizing power of the body against remaining lesions.—*Treatment of Genital Tuberculosis in Male, J. H. Cunningham (Boston), Surg., Gynec. & Obst., October, No. 4, 1916.*

The Waves of Improvement in Renal Tuberculosis.—Macedo has followed to date the history of three patients with a tuberculous process in the kidney, who refused to permit operative treatment, and their kidney trouble seemed to pass into a latent phase. The secondary bladder changes also subsided more or less completely. The phenolsulphonephthalein test and Ambard's uremic constant, however, show that the functioning of the kidney has not improved. The detailed findings from clinical examination repeated at various times during the year are given for each time in each case. The symptoms which have subsided are the pain in the lumbar region, abnormal frequency of micturition and hematuria. The kidney proper is not really sensitive and the local pain is from stretching of the capsule, etc. As the kidney shrivels the pain subsides. On the other hand, the polyuria persists constantly and with it the albuminuria and pyuria, the latter minute but constant, and tubercle bacilli are still found in the urine. These findings show that the improvement with advanced renal tuberculosis is deceptive. When a tuberculous process in the kidney has reached the "open" stage, we can never count on a spontaneous cure or on recovery under medical measures alone.—*Los periodos de mejoria en la tuberculosis renal, C. M. Macedo, Cronica Medica (Lima, Peru), XXXIII, No. 1.*

Iodin Treatment of Tuberculous Epididymitis.—In the case reported the solution of iodin and potassium iodid was injected directly into the fistula every few days and a subcutaneous injection made every day—all according to Durante's technic. The patient was thus given eighteen local and over 300 subcutaneous injections. When seen again six months later the young man was apparently in a most satisfactory, well nourished condition, the mucous membranes pink and the

fistula healed. The nodules on the epididymis were soft, not tender, and reduced in size and number.—*Caso di tubercolosi dell epididimo guarito con la cura alla Durante, A. Sofia, Gaz. degli Osped e delle clin, September 28, 1916.*

Tuberculosis of Female Genital Organs.—The article discusses the mode of entry of the tubercle bacilli into the internal genital organs, reviewing the literature on the subject and comparing clinical findings with the results of considerable personal research on guinea-pigs. H. injected tubercle bacilli into the vagina of the guinea-pigs or into the cornua of the uterus. All the data presented apparently disprove the possibility of ascent of tubercle bacilli against the course of the secretions. Even when implanted in a cornu of the uterus, the tuberculosis always spread in the distal direction.—*Mode of Invasion and of Spread of Tuberculosis in the Female Genital Organs. (Om Indgangsport og Udbrednings-maade af Tuberkulosen i det kvindelige Genitalsystem.) I. P. Hartmann, Hospitalstidende, November LIX, 1916.*

Fistula in Ano.—Göz has studied ninety-five cases of this affection in the Munich Surgical Clinic. In 43 per cent tuberculosis was in evidence as a factor. Division of the sphincter caused incontinence only when several times repeated. The rule is therefore to divide but once. Single incision with the thermocautery will cure 67 per cent of those treated, while repeated incisions give only 14 per cent of cure. The presence of tuberculosis aggravates the prognosis.—*Inaugural Dissertation, Tübingen, 1916.*

Thrombophlebitis in the Tuberculous.—Thrombophlebitis, as a complication in tuberculosis, has been observed seven times in 1400 cases at the Chicago Fresh Air Hospital. Three cases occurred in men and four in women. The four women were in the advanced stage. The lower saphenous was the site in five cases, both legs being involved. One woman (autopsy) had thrombosis of the inferior vena cava and of both iliac veins. One woman had phlebitis of the arm extending upward into the external jugular vein. This patient improved after leaving the hospital but died some months after. The other woman died with the phlebitis still active. One man is well, his pulmonary condition under control by artificial pneumothorax. The second man is an invalid but free of vein symptoms. The third man is convalescent from his pulmonary condition, his thrombosis manifest only through the thickened veins palpable under the skin. The case which came to autopsy was that of a married woman, aged 30, who was ill for 5 years and had signs of far advanced pulmonary disease. Bacilli could not be found in the thrombus by means of the carbol fuchsin stain, but with the pirin stain a few bacilli were found in the thrombus in the vena cava, and one bacillus in the intima of the iliac vein. With the Much stain, granules were found in many sections of the vena cava.—*Thrombophlebitis in the Tuberculous, with Autopsy, Ethan A. Gray, Medical Record, October 7, 1916.*

Pregnancy in the Tuberculous.—Norris reports 68 cases, and asserts that, except in the advanced cases of pregnancy, coexisting tuberculosis has little or no tendency to produce abortion, miscarriage, or premature labor. In about 20 per cent of mild, quiescent cases, however, and in 70 per cent of more advanced cases, exacerbations of the tuberculous process occur during pregnancy or the puerperium. Pregnancy should be avoided by tuberculous women unless the disorder is in the first stage and has been quiescent for at least two years. If, before the fifth month of pregnancy, evidences of activity of the disease appear, the uterus should be emptied, preferably by curettage during the first six or eight weeks and in later cases by vaginal hysterotomy. When pregnancy is thus interrupted, the prognosis is greatly improved, though amelioration of the lung condition is not positively insured. Before the fifth month of pregnancy, evacuation of the uterus, as soon as acute symptoms arise, leads to definite improvement in 65 to 70 per cent of cases, provided suitable after-treatment is applied. Intervention postponed a week or more from the onset of the exacerbation yields less satisfactory results. After the fifth month of pregnancy, expectant treatment is generally best. Labor should be made as easy as possible, the patient being rarely, if ever, allowed to go to term, but delivered by induction or premature labor two weeks before term. At labor, forceps or version is usually indicated. Throughout, hygienic and dietetic treatment should be applied; the patient should be carefully examined at regular and frequent intervals. Tuberculous mothers should not be allowed to nurse their infants, which should be especially guarded from infection.—*Pregnancy in the Tuberculous and Its Management, C. E. Norris, Am. Jour. of Obstetr., June, 1916.*

Ferran on the Bacteriology of Tuberculosis.—As is generally known, Koch's bacillus does not explain all the phenomena of tuberculosis, and especially does it fall short in connection with the problems of acquired immunization. Some authorities go so far as to state that the acid-fast bacillus of Koch is wholly devoid of either immunizing or curative powers. If there were not some natural immunizing forces in the body tuberculosis would in a short time destroy the race. Koch's bacillus cannot confer such protection. Ferran, the Spanish bacteriologist, insists that when tuberculosis arises *de novo* in the body, it is not caused by Koch's bacillus as such, although the power of the latter to cause inoculation tuberculosis is not called into question. According to Ferran, Koch's bacillus makes up but a small fraction of the complete biology of spontaneous tuberculosis, being merely the last step. Ferran says that in human beings conjugal infection but rarely occurs—the incidence varies from 7 to 22 per cent according to the material—and he does not understand how any spouse could escape infection if Koch's bacillus was the sole or even the chief agent of transmission.

Ferran holds that Koch's bacillus is naturally a saprophyte. He finds five varieties of bacteria present in tuberculosis, one of these representing the classical acid-fast bacillus of Koch. Literally it is the fourth term of an ascending mutation, due to adaptative evolution. A further form is that derived artificially from the tubercle bacillus. The Koch organism is by no means the same thing as the virus of tuberculosis. The artificial form is regressive, just as the three ascending forms are progressive, Koch's bacillus standing at the apex. The natural forms cause spontaneous tuberculosis but are not infectious from man to man. They are ubiquitous, in the air, soil, and water. If by any chance they enter the blood they give rise to a form of septicemia. If they gain access to the tissues, there is no immediate reaction, but instead a process of adaptation on the part of both bacterium and host, the latter obtaining a general immunization from the formation of antibodies in the blood, while the former tends to pass through mutations until Koch's bacillus is formed. If the latter stage is reached there has been no immunization—the individual is tuberculous; but in the majority of cases this is a local process admitting of spontaneous arrest, the subject is affected, but not infected with the disease.

Ferran's teachings apply to bacteriology in general and go far to fill a number of defects in present day teaching. According to Persano (*Rivista critica di clinica medica*), these views have excited much controversy. The opposition is based partly on the claims that vaccines and sera produced under Ferran's teachings have not yet led to practical results. Ferran's rejoinder to his critics is that his methods are in the experimental stage, but show encouraging results. Evidently several years must elapse before a final judgment regarding these claims can be formed.—*Editorial, Med. Rec., October 28, 1916.*

Culture Medium for Tubercle Bacillus.—Preparation of the medium: 1. Egg-white solution; dilute egg-white with ten parts of distilled water and shake thoroughly. To clear it, pass through a thin layer of cotton and then heat to 100°C. to hasten precipitation. Filter through paper. 2. Egg-yolk solution; dilute yolks with ten parts of water and stir well. Clarify by adding normal sodium hydroxid. Complete solution of the yolk is not desirable. The emulsion should be slightly turbid. To attain the proper degree of turbidity, 1 cc. of normal sodium hydroxid is usually added to each 100 cc. of the emulsion. The solution is heated to 100°C. and filtered. 3. Meat infusion; 500 gm. of finely chopped lean veal are covered with 1 liter of water containing 15 per cent of glycerin, allowed to infuse for twenty-four hours and filtered; 5 gm. of sodium chlorid are added, and the infusion heated to boiling. It is again filtered and then rendered plus 1 per cent alkaline. Place 300 cc. of the 10 per cent egg-white solution in a liter flask; 300 cc. of the 10 per cent egg-yolk solution in another flask; and 400 cc. of the meat infusion, to which is added 15 gm. of powdered agar-agar, in a third flask. Sterilize in autoclave at 15 pounds pressure for fifteen minutes. While hot, 1 cc. of a 1 per cent alcoholic solution of gentian violet is added to the broth agar. The contents of this flask are now poured into that containing the egg-white and then the egg-yolk is added. The whole is poured back and forth from this flask to another so as to insure thorough mixing and then it is tubed and slanted. The tubes are left in their slanted position for about seventy-two hours at room temperature until the contents are well set. The cotton plugs are then trimmed and flamed and the tubes sealed with corks. This medium presents the same smooth inoculating surface as ordinary agar slants, contains as much moisture, is quickly made and is rendered absolutely sterile.—*New Culture Medium for Tubercle Bacillus, W. W. Williams and W. Burdick (Denver), Jour. of Bacteriol., July, I, No. 4, 1916.*

Tubercle Bacilli in Breast Milk.—Five tuberculous women who had given birth to children while in Sea View Hospital were subjected to a total of 65 examinations. The milk was expressed from the breasts under aseptic precautions and bacilli were searched for both in a plain smear and in a smear prepared by the anti-formin method. None of the women had breast involvement; three were moderately advanced and two far advanced cases of pulmonary tuberculosis. All the specimens were negative except one which was once positive and once suspicious. This was a case of moderately advanced tuberculosis with bone complications and which left the hospital with no symptoms of active pulmonary disease and with the bone tuberculosis "arrested."—*Occurrence of Tubercle Bacilli in the Breast Milk of Tuberculous Women, S. L. Wang, Bull. Dept. Public Charities, New York City, October, 1916.*

The Relationship Between the Nervous System and Therapy in Pulmonary Tuberculosis.—The more important factor in the production of temperature is an interference with normal heat dissipation, rather than an unusually active heat production. In support of this theory P. offer the following facts: (1) The experimental introduction of protein produced a general sympathetic stimulation. (2) The manifestations of clinical toxemia were those of general sympathetic stimulation. (3) It had been noted on experimental animals that when protein was injected parenterally that there was a constriction of the vasomotors of the surface, as determined by the constriction of the vessels of the ear of the rabbit (Jona). (4) A rise of temperature was accompanied by other symptoms of toxemia and was accompanied by constriction of the superficial blood vessels, which, at times, manifested itself by a sensation of chilliness. (5) The action of toxins and adrenin was practically the same; both acted chiefly upon the nervous system. The introduction of adrenin caused a constriction of the superficial blood vessels, and a rise in body heat. From these facts he was led to believe that the rise in temperature which occurred in natural infections, or whenever a foreign protein of any type was introduced parenterally into the body of either man or animal, would not occur apart from their action through the sympathetic nervous system, which produced a general vasoconstriction of the superficial blood vessels, and interfered with the dissipation of heat. He was further convinced of this from the fact that emotional states, particularly the depressive emotions, such as those of fear, disappointment, anxiety, discontent, and worry, produced a general sympathetic stimulation; and that all of these depressive conditions were associated with a rise in temperature, at least in the tuberculous individual. Since sympathetic stimulation was produced by toxins, no matter what their source, we might find it in tuberculosis as a result of the absorption of protein from the bacillus itself, probably also as a result of the absorption of destroyed tissue and from any other bacteria that might complicate the process. While toxemia could not be caused by the depressive emotional states, yet these produced sympathetic stimulation and acted in the same manner as toxins; consequently, they must also be relieved the same as toxemia, if we would relieve the organisms from the inhibiting action of the sympathetic system.

Rest was an essential in treating toxemia. It was valuable whenever exercise increased the sympathetic stimulation by pouring forth into the tissues an increased amount of toxins. Thus we note that patients with rapid heart's action, poor appetite, deficient digestive activity, constipation and rise of temperature might experience an improvement, or even lose all of these symptoms under rest; and, as they disappeared, encouragement followed and a general improvement took place.

The influence of *food* might be greatly enhanced by relieving the sympathetic stimulation due to toxemia and depression. He usually noted that the appetite and digestion, and, therefore, nutrition of the patient improved when he was put at rest in the open air and given hope and encouragement.

There was a time when *exercise* had an important and beneficial influence upon the patient. Exercise called for increased metabolism. Increased metabolism was met by increased food intake; consequently, after toxemia had passed and the patient could exercise without producing toxic symptoms we could greatly improve his nutrition by permitting exercise.

The effect of *baths*, either air, sun, or water baths, was exerted through the nerve endings in the skin, and extended to every cell of the body. If the reactivity of the skin was well cared for, as it might be through the stimulating effect of water, light, or air, upon it, the vasomotor tone was improved, the metabolic activity of the patient's cells was hastened, and the elimination of heat, moisture, and various toxic products was hastened.—*The Relationship between the Nervous System and Therapeutics in Pulmonary Tuberculosis, F. M. Pottenger, N. Y. Med. Jour., November 11, 1916.*

A CASE OF INTRATHORACIC CYST WITH CALCIFICATION

GEORGE J. HEUER

From the Surgical Service of the Johns Hopkins Hospital

Calcification occurring in association with conditions within the thoracic cavity is not uncommon. It is found, for example, in chronic suppurative processes in the pleura, of tuberculous, pneumococcic, or pyogenic origin; in pulmonary tuberculosis; in haemorrhages into the pleural cavity; and in the walls of dermoid and echinococcus cysts. Were it not for the unusual extent of the calcification and the clinical and pathological interest attaching to the condition in the present instance, we should hesitate to report a single case.

The patient, a colored laborer, fifty-three years of age, entered the Johns Hopkins Hospital in the service of Dr. Janeway, October 12, 1916. He complained of cough, pain in the right side, and shortness of breath. His family history was unimportant. He had had typhoid fever and pneumonia involving the left lung thirty-two years ago; a Neisser infection and a genital lesion thirty years ago, but not followed by secondary or tertiary luetic manifestations; and a perforation of his nasal septum, the result of his occupation in an acid factory, fifteen years ago. With the exception of pneumonia, he gave on admission no history of symptoms referable to his respiratory tract previous to the onset of his present illness. Questioned since his recovery from operation, however, he states that he now realizes that he had some "misery" in his side and shortness of breath on exertion for an indefinite period. He had been married twice. His first wife died of tuberculosis at the age of thirty-eight. His second wife died two years ago of cancer of the stomach.

Present illness. One morning four weeks before admission, while shifting bags of fertilizer each weighing 300 pounds, he had a sudden severe pain in the right side of his chest, with cough and shortness of breath. The symptoms were so severe that he was compelled to stop work, but was able to walk to a doctor's office. The physician advised rest in bed, and he remained in bed until his admission to the hospital. The cough has persisted while the pain and shortness of breath have been less troublesome since he has been lying quietly in bed.

Physical examination. A very large, well nourished, muscular man, apparently not seriously ill. The general physical examination, with the exception of that pertaining to his right thorax, was entirely negative, and need not be included. His temperature during the period he was under observation varied between normal and 100°, with an almost daily rise in the afternoon to 99° or 99.2°.

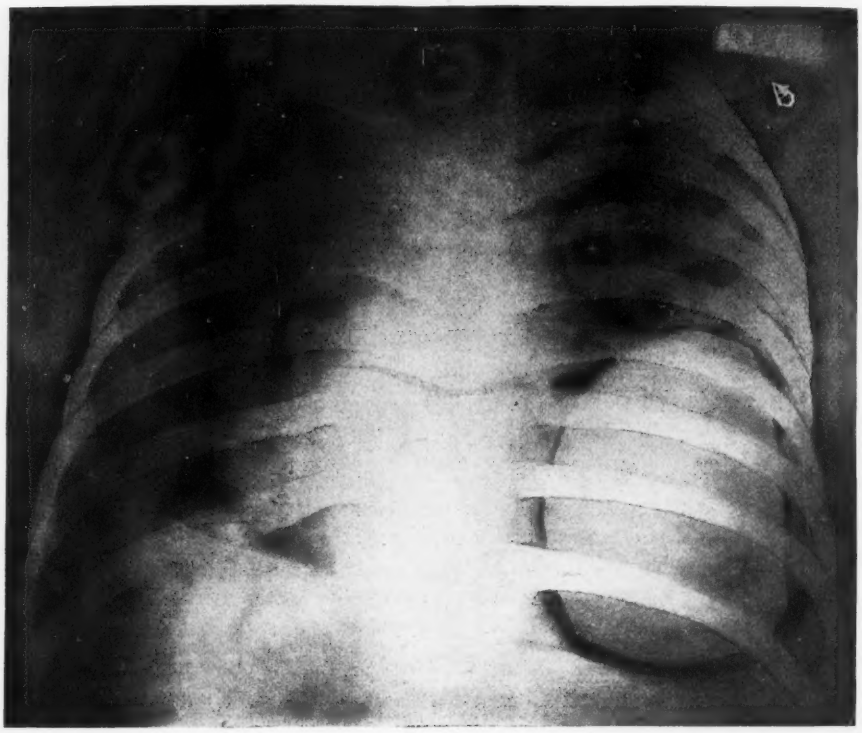

FIG. 1. ROENTGENOGRAM OF PATIENT IN THE PRONE POSITION

Showing the outline of the mass in the right thorax

Right thorax. Inspection: the right side of the chest is somewhat flattened over the right upper front. There is diminution in expansion of the entire right side. Palpation: vocal fremitus is moderately increased about the apical region in front and behind. It is diminished, but still present, over the lower axillary and subscapular regions. Percussion: the percussion note and resistance are distinctly increased above the clavicle and over the supraspinous fossa. With the patient in the erect position, flatness begins at the upper margin of the fifth rib, and in the recumbent position, in the middle of the sixth in-

terspace. Behind, the flatness begins at the level of the angle of the scapula, and continues downward. Beginning just below the level of the line of flatness on the right side, there is at least a suggestion of a Grocco's sign on the left. Auscultation: on auscultation the breath sounds are enfeebled at the

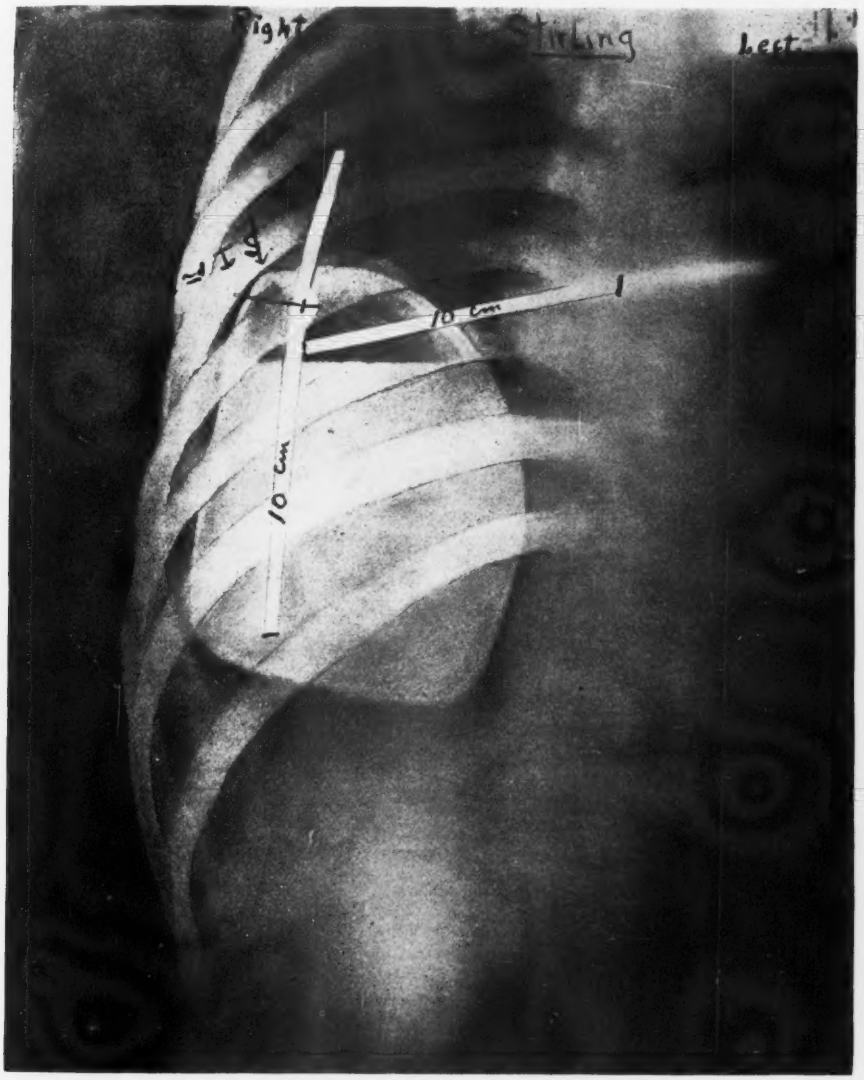

FIG. 2. TELEOROENTGENOGRAM OF THE PATIENT IN THE ERECT POSITION

Showing the mass containing fluid and air. A suggestive defect in the wall may be seen at the upper outer pole.

apex in front and behind. There is an occasional fine bubbling râle on inspiration. Below the line of flatness in front and behind, the breath sounds and voice sounds are enfeebled and the vocal resonance is diminished. No pleuritic friction rub is audible.

The patient was repeatedly examined by various members of the medical staff, and in general the above physical findings were corroborated. The diagnosis on physical examination alone was a right-sided pleurisy with effusion, of uncertain origin. A thoracentesis was attempted before a Roentgenogram had been made. The exploring needle introduced at the eighth interspace behind met with resistance at a certain depth and could not be introduced farther. No attempt was made to force the needle beyond the obstruction. The procedure resulted in a "dry tap."

Roentgenological examination. Roentgenograms of the chest explained the cause of the obstruction to the exploring needle and were of the greatest aid in the diagnosis of the condition. The Roentgenograms (see figs. 1 and 2) show in the right thoracic cavity a perfectly circumscribed shadow with remarkably clear outlines. This shadow in general is pyramidal in shape, its base flattened and resting upon the diaphragm, its apex rounded and extending 11.5 cm. upward into the thoracic cavity. Mesially, the structure is in contact with the mediastinum, laterally it approaches to within about 1 cm. of the rib margin. The wall of the structure varies from 1 to 2.5 mm. in thickness. With the patient prone, the contents of this apparently cystic structure cast a shadow of uniform density (fig. 1). In an erect position (fig. 2) they separate into two layers, a lower, consisting of fluid and casting a definite shadow; and an upper, consisting of air and represented by an absence of shadow. Close examination of the wall of the structure shows apparently a crack at its upper outer pole. Examined stereoscopically, the mass appears to lie nearer the anterior thoracic wall than the posterior.

Laboratory findings. Red blood cells, 3,552,000; haemoglobin, 60 per cent; white blood cells, 7240. Differential count, polymorphonuclears, 66 per cent; eosinophiles, 0.3 per cent; basophiles, 0.3 per cent.; small mononuclears, 13 per cent; large mononuclears, 14 per cent; transitionals, 6.3 per cent. Wassermann reaction (blood and spinal fluid) was negative. Urine examination showed no abnormality. Sputum, the daily amount varied. On some occasions during paroxysms of coughing the patient expectorated a cupful of yellowish or reddish brown seropurulent sputum. It had a slightly foul odor and contained numerous cholesterin crystals, numerous pus and epithelial cells, a few red blood corpuscles, many irregular large and small granular cells containing black pigment dots, and an occasional structure 20 micra in diameter which when stained proved to be an organism containing an ectosarc and an endosarc and provided with a unipolar group of cilia (probably related to Rhizomastigina). Repeated search and digestion studies failed to

show hooklets. Hairs were never found and tubercle bacilli could not be demonstrated.

Summary and discussion. The patient was a man who up to the day of onset of his illness had performed the heaviest labor and was unaware of the condition in his chest until a physical strain gave rise to pain, cough and shortness of breath. He presented the physical signs of pleurisy with effusion. In an attempt to confirm the diagnosis by thoracentesis, the exploratory needle met with resistance such as might be offered by a bony structure. The Roentgenograms made possible a diagnosis which could not have been reached by physical examination alone. From these it was evident that there was present a cystic condition with complete calcification of the cyst wall, and that the cyst contents consisted of fluid and air. That there was a communication between the cyst and the lung was clinically evident because of the periodic expectoration of large amounts of sputum. The Roentgenogram apparently showed this point of communication. Following the Roentgenological studies diagnostic efforts were directed toward establishing the nature of the cystic condition. There was no evidence of tuberculosis, no eosinophilia and no characteristic findings in the sputum, such as hooklets or hairs. Etiologically, there was no history of pulmonary affections, of trauma of the chest or of intimate association with dogs. A positive diagnosis could not be made. The diagnoses suggested were encapsulated empyema, haematoma of the thorax, echinococcus cyst and dermoid cyst.

Operation. November 8, 1916. Under ether anesthesia, through a long incision, about 20 cm. of the ninth rib were excised. The parietal pleura was stripped away from the thoracic wall so as to allow satisfactory inspection and palpation of the lesion before opening the pleural cavity. On palpation through the detached pleura, it was at once evident that an extremely hard mass lay directly underneath and was firmly attached to it. A horizontal incision was made through the parietal plura and the exposed portion of the mass examined. Its presenting surface was whitish in color and of bony hardness. An attempt was made to find a layer of cleavage between it and the parietal pleura, but this was entirely absent over its lateral and anterior aspects. It was necessary to cut across the adhesions with scissors and knife, and after this had been done for a considerable distance in all directions, the anterolateral surface of the shell was largely exposed.

The operator was a little doubtful as to how to continue, but having begun to enucleate the mass he kept on with this procedure. As previously noted, it was necessary to cut between the adherent pleura and the outer wall of the mass over its entire anterolateral aspect. Having freed this surface, the operator next explored that in contact with the diaphragm. Here the adhesions became less dense, so that it was easily possible to strip away the

diaphragm from the inferior surface of the mass. The operator then explored the mesial wall of the cyst, and found dense adhesions between it and the anterior mediastinum. These were cut with knife or scissors until this aspect of the mass was freed. Unfortunately, at the lower mesial aspect of the cyst the operator penetrated its wall, and there followed a discharge of a small amount of a thin yellowish purulent material and some air bubbles. The operator packed some gauze into this opening and temporarily left this region.

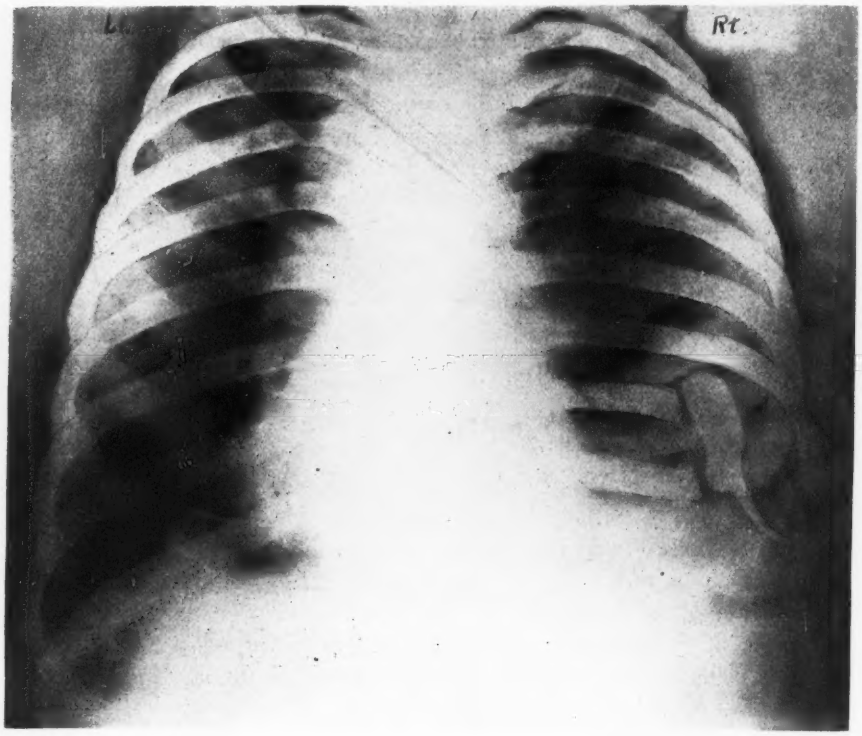

FIG. 3. ROENTGENOGRAM OF PATIENT TAKEN TWO AND A HALF MONTHS AFTER OPERATION

The size of the cavity represented by the shadow cast by the bismuth paste filling it.

Inserting his hand between the inferior aspect of the mass and the diaphragm, he found that the posterior and posteromesial surfaces of the structure could be stripped away from the overlying lung with remarkable ease, so that in a few moments this region was freed from what subsequently proved to be the concave surface of the lower lobe of the lung. The operator then proceeded to free the lateral aspect of the shell which also was densely adherent to the

parietal pleura. The freeing of the upper pole of the mass gave especial trouble, and before this was accomplished it seemed necessary to enlarge the operative field by the resection of about 15 cm. of the eighth rib. A narrow strip of lung tissue adherent to the upper pole was cut across and left upon the cyst wall. While freeing this part of the mass there occurred a spontaneous escape of a small quantity of cyst contents and air bubbles, and it was

FIG. 4. ANTEROLATERAL VIEW OF THE MASS

Showing roughened surface which was densely adherent to the pleura.

assumed that the communication between the cyst and the lung was at this point. The last step in the enucleation of the mass was the freeing of its mesial and anterior walls from the mediastinum. It was found that here the bony shell was not complete but was in part formed of dense fibrous tissue. By its division the cyst was opened widely and the major part of its contents escaped. The shell was finally removed in toto. There was no

haemorrhage throughout the entire procedure; indeed, it was not necessary to tie a single blood vessel.

After the removal of the mass, an enormous cavity remained, the inferior surface of which was formed by the diaphragm, the mesial surface by the mediastinum, the upper and posterior surface by the concave surface of the lower lobe of the lung, and the anterolateral surface by the thoracic wall. The right side of the heart lay under the mesial surface of the mass. It was

FIG. 5. POSTEROLATERAL VIEW OF THE MASS

Rounded surface to the left in the picture is smooth and quite free from adhesions.

noted that the visceral pleura of the lung in contact with the mass was not thickened. No attempt was made to obliterate the large cavity left after the removal of the mass. It was drained with a single large rubber tube. The wound was closed in layers.

Post-operative history. The patient left the operating table in good condition. There were no post-operative complications other than a slight wound infection. For three days the temperature varied between 99.5° and

100.2° and then came to normal. Bismuth injections showed a rapid obliteration of the cavity in the right thorax (fig. 3). At the present time, March 1, 1917, the cavity is entirely obliterated, the wound healed and the patient apparently perfectly well.

EXAMINATION OF SPECIMENS

(See figs. 4, 5 and 6)

FIG. 6. MESIAL SURFACE OF THE MASS

Showing the defect in its wall.

The calcified mass lay within the pleural cavity, not within the lung. The specimen consists of a roughly pyramidal calcified shell which measures 13 cm. in its greatest horizontal diameter, 11.5 cm. in its greatest vertical height, and 7 cm. in its anteroposterior diameter. Its inferior surface, which rests upon the diaphragm, is flattened and slopes slightly upward and backward, its anterior surface is slightly flattened, while its upper and posterior surfaces are quite spherical. Its wall is complete with the exception of a roughly circular defect

about 4 cm. in diameter (fig. 6). The anterolateral surface of the shell is roughened and covered by dense fibrous tissue while its apex and posterior curved aspect are perfectly smooth. On examination it is quite evident that the entire shell, with the exception of the defect previously noted, is made up of calcified tissue measuring 1 to 2 mm. in thickness. Its inner surface is roughened and covered by a soft putty-like sediment 2 to 3 mm. in thickness. Microscopic sections show that this sediment is made up of detritus in which no structure which might give a clue to the nature of the condition can be found. Sections of the wall of the cyst show calcification and not new bone formation.

Complete studies of the cyst contents and of the sediment were made by Dr. Guthrie. They failed to establish the nature of the condition.

Our search of the literature for reports or Roentgenographic reproductions of similar cases has thus far proved unavailing, but has indicated that extensive calcification in thoracic conditions is not uncommon. Most frequently it has been found in association with dermoid cyst, echinococcus cyst, infections of the pleura, haematomata of the thorax and tumors. We will briefly discuss these conditions with reference to our case.

DERMOID CYST

There are a number of collected series of cases in the literature. We have consulted those of Roger Morris (1), who collected fifty-seven cases in 1905; of Dangschat (2), who collected forty-four cases in 1903; and of Pohl (3), who brought this number up to fifty-two in 1914. Dermoid cysts arise in the mediastinum and vary in size from that of a walnut to that of a child's head. The small tumors remain in the mediastinum while the large ones may extend outward into the pleural cavity. A few have been described occupying the right base. In shape they are usually spherical, but may be flattened when in contact with the diaphragm. The cyst wall may be very thin or quite thick, and is invariably adherent to surrounding structures, most commonly to the pleura. Partial calcification of the cyst wall has occurred in a number of cases (four in Morris's series) but not to such an extent as in our case. In one-half of the cases reported the cyst has perforated into a bronchus and therefore not only infection but the presence of air in the cyst has modified the character of the cyst contents. Cholesterin crystals are a common finding; and in the majority of cases the presence

of hair, epidermis, cartilage, bone, etc., permits a positive diagnosis. In the epidermoids, however, the cyst contents may consist merely of fluid and cellular detritus (the result of desquamtion of epithelial cells) and quite fail to establish a positive diagnosis.

HYDATID OR ECHINOCOCCUS CYSTS

From 10 to 19 per cent of cases of hydatid disease occur in the lungs. Robust health is not incompatible with the disease until some complication, such as perforation into a bronchus, occurs. While the distinction between primary hydatids of the lung and primary hydatids of the pleura is not made by some authors, who assume that the pleural infection is secondary to that of the lungs, there is in the literature a number of collected series of cases of primary hydatid disease of the pleura. Cary and Lyon (4) have collected forty cases and French observers have described the condition under the term "pachy-pleurite echinococcique." Vincent (5) describes cysts within the pleura, rather frequently at the right base, which in their growth have pushed the base of the lung upward. Some assume a pyramidal shape due to the flattening of the surface in contact with the diaphragm. The cyst wall varies in thickness and is characteristically laminated, but may be hyaline. It casts a definite shadow in the Roentgenogram which is more clearly defined when, as sometimes occurs, it has undergone extensive calcification. Rupture of the cyst into a bronchus is of frequent occurrence, resulting in a secondary infection of the cyst contents and in the presence of air in the cyst. The presence of scolices and hooklets in the cyst contents often establishes the diagnosis but frequently prolonged search does not reveal them. Nothing characteristic may be found when the cysts have perished and undergone complete disintegration. Cholesterin crystals are common. Eosinophilia and the complement fixation test may be negative.

The X-ray has proved of the greatest value in the diagnosis of this condition. Roentgenograms almost identical with figure 2 are reproduced in the literature (6) and have been considered characteristic of thoracic hydatids which have perforated into a bronchus. The characteristic picture is spoken of as McCormick's sign, but that it need not be peculiar to hydatids is evident.

INFECTIOUS PROCESSES IN THE PLEURA

The infections may become quiescent and the thickened pleura undergo extensive calcification. Tuffier (7) describes a case of complete calcification of the pleura in a patient with chronic empyema, and similar observations have been made by other authors. A striking

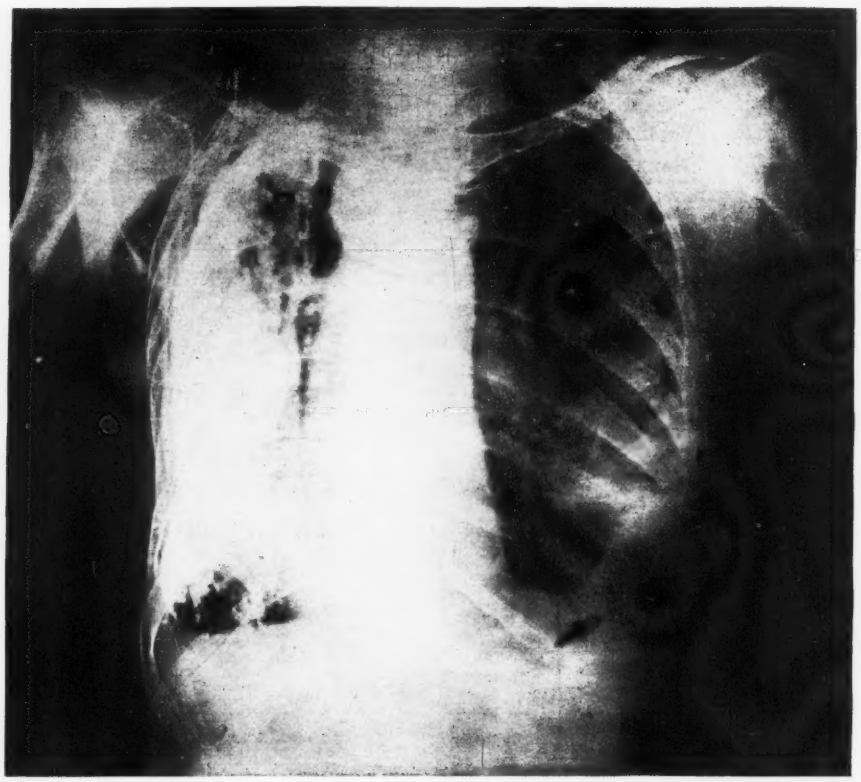

FIG. 7. ROENTGENOGRAM OF PATIENT

Showing complete calcification of the pleura (service of Dr. Janeway)

example of almost complete calcification of the pleura (fig. 7) unsuspected until a Roentgenogram of the chest had been made, and confirmed by exploratory thoracentesis, occurred recently in the service of Dr. Janeway. Pleural calcification presents itself in many forms. It may involve the greater part of or even the entire pleura, with the formation of a cavity, or, as it has been described, a "pseudo-pleural

cyst." It may occur in the form of large plaques, of stalactites, or of pseudosequestra. Most frequently it is found about the diaphragm, about the costo-diaphragmatic angle, and along the spine. In the cases in the literature calcification of the pleura has given rise to few symptoms, and in most instances has been an accidental autopsy finding. It has, however, occasionally been discovered in patients during a thoracentesis. Pathological examination of material removed at operation or discovered at autopsy has shown that the condition is the result of calcification, not of ossification.

HEMORRHAGE INTO THE PLEURA

This may occur following trauma, such as injury to the ribs and penetrating wounds. A haemorrhagic pleurisy may occur in association with tuberculosis, cirrhosis, and malignant tumors. There is described in the literature a primary haemorrhagic condition of the pleura called "haemorrhagic pachypleuritis" associated with the formation of a neomembrane and with thickened pleura. By some this condition is believed to be of tuberculous origin; by others it has been found in the absence of this disease, and comparable to the condition of haemorrhagic pachymeningitis. Large haemorrhagic cysts, or haematomata of the pleura, have been found as an unexpected autopsy finding. In general, calcification is common in haematomata, but from our survey of the literature it is rather rare in haematomata of the thorax. A case of Tuffier's (7) may be cited as an example, complicated, it is true, by infection. The patient was a man who, while carrying a heavy load, fell, striking his upper left thorax. He had severe pain, sufficient to warrant his entrance to the hospital. Tuffier found and incised an infected hematoma of the thorax over the left fifth rib. The wound healed in ten days. Three years later the patient after sudden exertion had marked pain in the left chest, and again entered the hospital. While under observation in the hospital, a fistula formed at the site of the previous operation. Tuffier resected a rib and on exploration found a large cyst-like structure with extensive calcification of its wall, presumably an old haematoma, the result of the previous injury. There is no record of an X-ray examination in this case.

Bony tumors of the pleura. This condition probably bears no relation to our case. Often of metastatic origin, these tumors may be quite

extensive and involve the adjacent surfaces of the visceral and parietal pleurae with the formation of a cyst-like cavity between them. The lesion, however, is a true bony one, not the result of calcification.

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RENAL FUNCTION IN PULMONARY TUBERCULOSIS

ELMER H. FUNK

From the Department of Medicine of the Jefferson Medical College, Philadelphia, Pennsylvania

The ultimate outcome in pulmonary tuberculosis is dependent so often upon the structural integrity and functional efficiency of organs other than the lungs, that a study of them is of importance. In spite of the fact that much has been written, there still exists a vast field for investigation along these lines. The need of such studies is well illustrated by reference to the meagreness of our knowledge of the gastric function in this disease and the recognized clinical importance of the digestion. Mohler and the writer (1) recently reviewed the literature upon the subject and were impressed with the absence of any systematic or accurate observation upon gastric function, although repeated references are made to the importance of good digestion in the prognosis. The present study was undertaken to determine the relation of renal function to prognosis in patients with pulmonary tuberculosis. It was thought that such a study, comprising as it does an investigation of the eliminative power of the kidney, would be particularly interesting in view of the frequent structural changes which occur in these organs as the disease progresses. The phenolsulphonephthalein test of Rowntree and Geraghty was applied according to the technique originally described by these observers. This test was selected in preference to the blood nitrogen, urea nitrogen, renal test diet, etc., because of its simplicity and general utility in a large series of observations. In the early part of our work an effort was made to estimate the glomerular and tubular functions by the differential tests of Schlayer and his co-workers, but the results were at such variance with the other data that conclusions as to specific functions seemed hazardous. Twenty-four patients with incipient, and 114 patients with advanced tuberculous lesions in the lungs were selected. An ordinary chemical and microscopical examination of the urine and a phthalein test were made upon admission and at intervals afterward, and in a few cases these observations were followed to autopsy.

STRUCTURAL CHANGES IN THE KIDNEYS

Among the 24 incipient cases the urine was examined from two to six times with the result that a trace of albumin was found in three patients, casts were absent in all the specimens and the phthalein test was above 60 with the exception of one, in which it was 58. The average for the group was 73. Structural changes, therefore, could not be demonstrated and the eliminative power was apparently normal. Most of these cases could not be followed up because they were sent to sanatoria or failed ro report back to the hospital for study.

Among the 114 advanced cases which were admitted to the wards, the ordinary urine examination upon admission revealed structural changes in a large number and the relation of the urinary findings to the outcome is noted in Chart I.

CHART I

Analysis of mortality rate during six months with reference to the urine findings upon admission

URINE FINDINGS	NUMBER OF PATIENTS	NUMBER OF DEATHS	MORTALITY RATE FOR THE GROUP
			per cent
No albumin or casts	52 (45.6 per cent)	8	15.4
Albumin	22 (19.3 per cent)	7	32.0
Albumin and casts	40 (35.1 per cent)	16	40.0
Total	114	31	

Among this group of 114 patients the mortality was 31. An analysis of the deaths reveals that 8 occurred among 52 patients with no albumin or casts, a mortality rate of 15.4 per cent; 7 among 22 patients with albumin only, or a mortality rate of 32 per cent; 16 among 40 cases with albumin and casts, or a mortality rate of 40 per cent. It is suggested that the presence of albumin or of albumin and casts increases materially the gravity of the prognosis. The examination of the urine at stated intervals after admission showed that in those cases proceeding to an unfavorable termination the presence of albumin and casts became more frequent. Analyzing the 31 deaths further upon this basis it was found that the group having no albumin upon admission was reduced from 8 to 3; the group having albumin only, from 7 to 5; while those with albumin and casts increased from 16 to 23. Expressed in another way, the figures of the following chart show that among 31 patients dying with advanced pulmonary tuberculosis, 9.7

CHART II

Analysis of thirty-one deaths with reference to urine findings upon admission and afterward

URINE FINDINGS	UPON ADMISSION	AFTER ADMISSION	PERCENTAGE OF PATIENTS SHOWING URINARY CHANGES BEFORE DEATH
	deaths	*deaths*	*per cent*
No albumin or casts	8	3	9.7
Albumin	7	5	16.1
Albumin or casts	16	23	74.2
Total	31	31	100

per cent showed no albumin, 16.1 per cent showed albumin only, and 74.2 per cent albumin and casts within six months of death.

Structural changes in the kidney at autopsy are frequently demonstrable. In a study which was characterized by a most painstaking histological examination, Walsh (2) found the following renal lesions among 100 cases of pulmonary tuberculosis: acute parenchymatous nephritis, 36; chronic parenchymatous nephritis, 33; diffuse nephritis, 4; interstitial nephritis, 14; amyloid degeneration, 5; cloudy swelling, 7; passive congestion, 1. Walsh states that it is probable that the cloudy swelling and acute nephritis are due to terminal infection and bear no relation to the tuberculosis, but that the 56 kidneys showing a chronic condition might have been influenced by the tuberculosis. Chronic diffuse nephritis is the common type of nephritis in patients with pulmonary tuberculosis and it is probably more frequently found in this disease than in any other. This is not strange when we recall the prolonged toxemia attending the disease and the added harmful influence of the multiple superimposed infections which attack the majority of patients and last for weeks or months, and sometimes years.

Our own pathological studies of the kidney confirm those of Walsh. Among 81 necropsies the renal lesion, other than tuberculosis, was noted as follows (the percentages allow of comparison with Walsh's 100 cases): acute or subacute diffuse nephritis, 30 (37 per cent); chronic diffuse nephritis, 33 (40 per cent); chronic interstitial nephritis, 6 (7 per cent); cloudy swelling, 4 (5 per cent); passive congestion, 8 (10 per cent); hyperemia, 1 (1 per cent). With regard to the relation of the nephritis to the presence of tubercles in the kidney Walsh found tubercles in 55 of his 100 cases. Their association with all the different forms of

nephritis occurred in such numbers that they could not be considered to have any influence upon the nephritis; and reversely, the nephritis little or no influence on the development of the tubercles.

FUNCTIONAL EFFICIENCY OF THE KIDNEYS

I have stated that among the 24 incipient cases there was no evidence of impairment of the eliminative power of the kidney. The average phthalein test was 73. Among the 114 cases with advanced lesions in the lungs the renal function upon admission was below that of the incipient cases, but the impairment was certainly not marked in the average case, either of the males or females.

If we consider an elimination of from 60 to 80 per cent phthalein in two hours as normal, the renal function in the average advanced case upon admission is about the minimum normal point. When we compare the urine findings with the phthalein test we find that although a reduction in function below what is considered normal occurs when structural changes are present, the reduction is not marked.

CHART III

Renal function upon admission (advanced cases)

	PATIENTS	MALES	FEMALES
Number	114	78	36
Average age	36.8 years	38.1 years	32.5 years
Average phthalein, first hour	43.4 per cent	41.9 per cent	46.3 per cent
Average phthalein, second hour	17.3 per cent	17.2 per cent	17.5 per cent
Average phthalein for two hours	60.7 per cent	59.1 per cent	63.8 per cent

By comparing Chart I with Chart IV it will be noted that the relationship between the structural changes in the kidney and the mortality is more evident than the reduction in renal function. We feel warranted in assuming that, although structural changes in the kidney occur with increasing frequency as pulmonary tuberculosis advances, and are of significance prognostically, the eliminative power of the kidney remains fairly good, even in the advanced stages. It is only in the last few days of life that renal function is materially reduced, and then as the result of terminal changes (Chart VI). In a certain number of cases the function may remain fairly good even in the last hours (Chart V). Unfortunately we were unable to follow the renal

CHART IV

Comparison of urine findings and phthalein test upon admission (advanced cases)

PERCENTAGE OF CASES IN WHICH URINE CONTAINED	PHTHALEIN ELIMINATION		
	1 hour	2 hours	Total
	per cent	*per cent*	*per cent*
No albumin or casts, 45.6 per cent	46.4	17.4	63.8
Albumin, 19.3 per cent	46.0	18.9	64.9
Albumin and casts, 35.1 per cent	37.7	16.0	53.7

function to the end in all the patients who died. The following charts, V to IX, illustrate our experience in a few instances in which such a study was possible.

CASE HISTORIES

E. L., female, 32 years, houseworker (see Chart V)

Present illness began eighteen months ago with cough and pain in chest followed by expectoration, progressive loss of weight and strength. Haemoptysis and night sweats. Upon admission the physical examination revealed advanced pulmonary tuberculosis with bilateral cavity formation. Sputum positive for tubercle bacilli. Urine negative upon admission. About one month afterward urine showed trace of albumin and casts. These findings continued until death. The renal function upon admission was good and continued so until the last month of life, when it dropped to around 50 per cent, and four days before death it was 44 per cent. The patient died from progressive asthenia and, although the autopsy revealed a distinct renal lesion, the functional efficiency was not sufficiently impaired to be a definite factor in the production of death. There were no clinical manifestations of renal insufficiency. The kidneys removed at autopsy were studied histologically by Dr. E. D. Funk who reports as follows:

The capsule shows a relative amount of thickening with some enlargement of the subcapsular capillaries. The parenchyma shows the greatest change in that the epithelial cells lining the tubules are greatly swollen, granular, fragmenting, and desquamating. This change is not, however, altogether general, but more or less patchy. A number of casts are found within the greatly narrowed lumina of the tubules. The interstitial tissue is not appreciably increased. The vessels everywhere are well dilated and filled with red cells. The glomeruli show no noteworthy changes except for an occasional fibroid one.

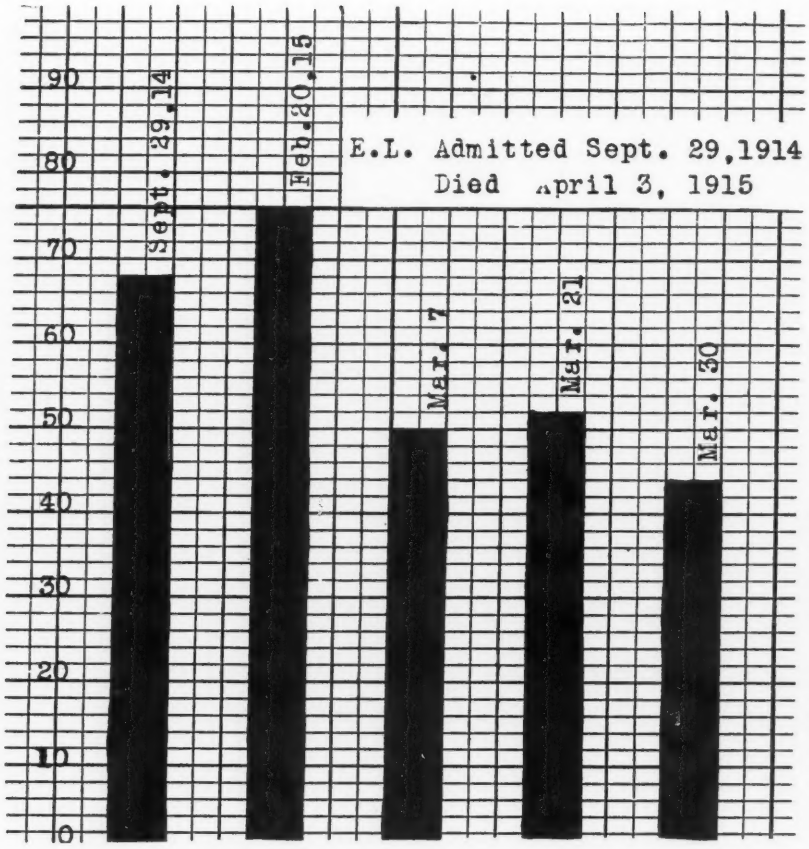

CHART V

L. N., female, 35 years, housework and child's nurse (see Chart VI)

Present illness began six months ago following an attack of "grippe" with cough and scant expectoration. Following this increasing expectoration, one attack of haemoptysis, hoarseness at times, progressive loss of weight and strength and night sweats. The physical examination upon admission revealed advanced caseous tuberculosis with bilateral cavity formation. Sputum contained numerous tubercle bacilli. Urine upon admission (in July 1914) was negative, in August, a trace of albumin appeared. In December and thereafter to March, 1915, increasing quantities of albumin to marked cloud and casts were present. There was slight pretibial oedema. The renal function, excellent upon admission, became reduced only during the last

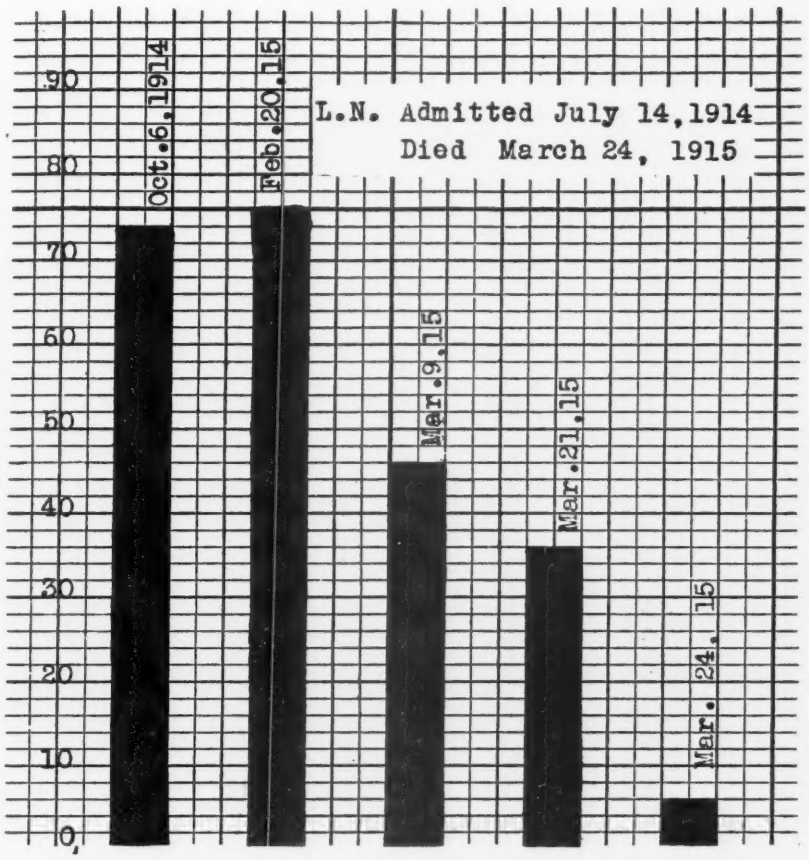

CHART VI

month of life, dropping to 36 three days before death, and to 6 upon the day of death. The histological examination of the kidneys by Dr. E. D. Funk revealed the following:

The capsule shows no appreciable increase in amount. The interstitial tissue, in parts, is increased but not to any great extent. In general, the parenchyma shows the greatest change. The epithelial cells of the tubules are greatly swollen so as to almost occlude the lumina. These cells, in places, have so greatly degenerated and desquamated that there is left but the lining membrane. Many of the tubules contain casts. The blood vessels are moderately filled with blood. None of the vessels show any degree of sclerosis. The glomeruli show no great change except for the deposit of amyloid material in the walls of the vessels.

Diagnosis. Chronic diffuse nephritis with marked parenchymatous degeneration.

S. L., male, 50 years, cement and stone mason (see Chart VII)

Present illness began four months ago with a "cold," with distressing cough and profuse expectoration which have continued. Dyspnoea at all times. Hoarseness. Progressive loss of weight and strength. Physical examination upon admission revealed advanced fibroid pulmonary tuberculosis with

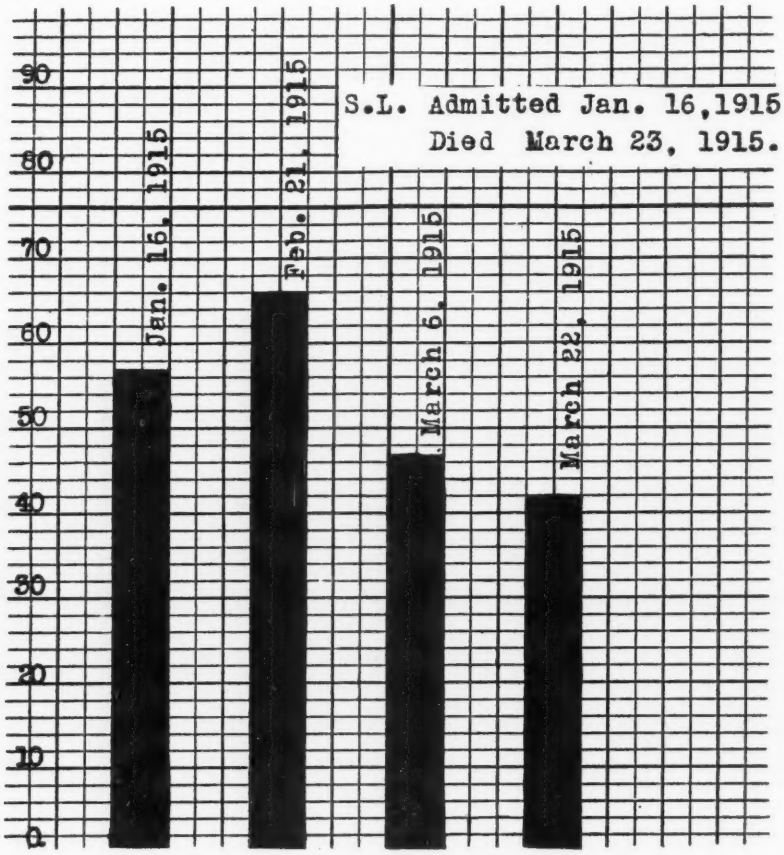

CHART VII

recent extension. Distinct clubbing and incurvation of nails. No uraemic symptoms or oedema at any time. Sputum positive for tubercle bacilli. Urine on admission and afterward revealed traces of albumin and a few granular casts at each examination. Renal function reduced as end approached, but at no times seriously impaired. Death due to progressive weakness.

J. K., male, 50 years, laborer in gas works (see Chart VIII)

Present illness began four years ago with pleurisy. Every winter a cough which now persists. Recurrent hoarseness. Expectoration. No haemoptysis. Loss of about forty pounds in weight since onset. Night sweats. Physical examination upon admission revealed moderately advanced pulmonary

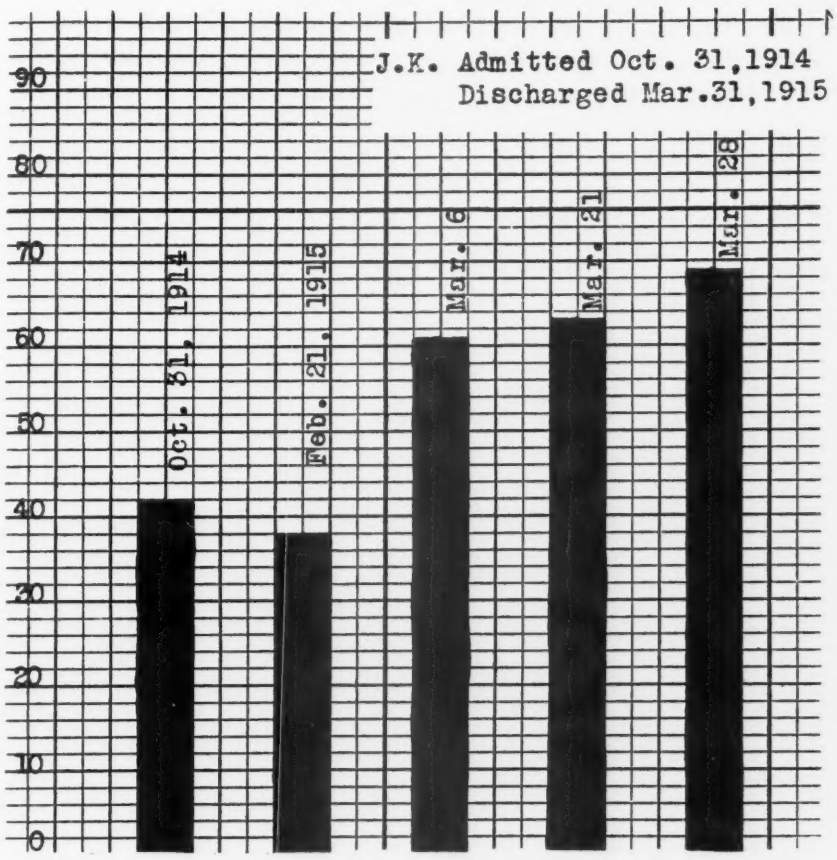

CHART VIII

tuberculosis with softening at right apex. The sputum was positive for tubercle bacilli. Urine examinations made every two weeks revealed at times traces of albumin and a few hyaline and granular casts. When under observation the general condition of the patient improved considerably. The pulmonary lesion became quiescent and incidentally the renal function improved.

D. S., male, 18 years, errand boy (see Chart IX)

Present illness began seven months ago with cough, expectoration and weakness. Severe attacks of blood spitting. Hoarseness, loss of appetite, loss of weight and strength. Physical examination upon admission revealed extensive pulmonary tuberculosis with bilateral cavity formation and mitral

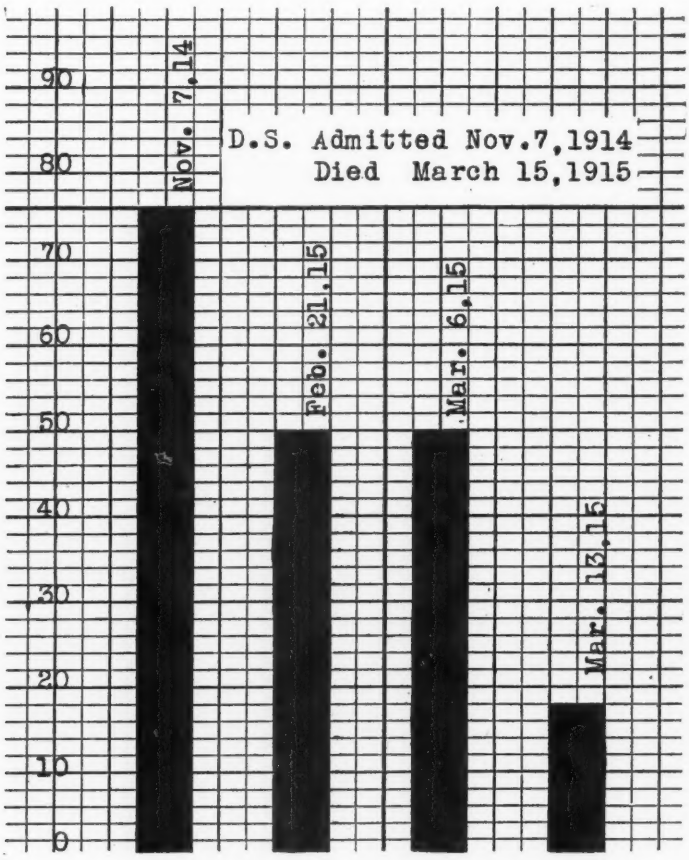

CHART IX

insufficiency with threatened cardiac decompensation. Sputum positive for tubercle bacilli. Urine upon admission was negative and phthalein elimination 76. Patient began to get weaker and developed evidences of failing heart with dyspnoea, oedema of face and ankles, albumin and casts. The phthalein elimination dropped to 50 and continued thereabouts until the

week before death, when an examination showed 18 per cent. During the last two days he was markedly cyanotic and somnolent. Death due to tuberculosis and cardiac failure with reduction in renal function in the last few days.

J. P., male, 27 years, day laborer out of doors (see Chart X), pleurisy five years ago

Present illness began six months ago with pain in right side of chest, and sweats. Cough, expectoration, loss of weight and strength. Physical examination upon admission revealed tuberculous laryngitis and advanced

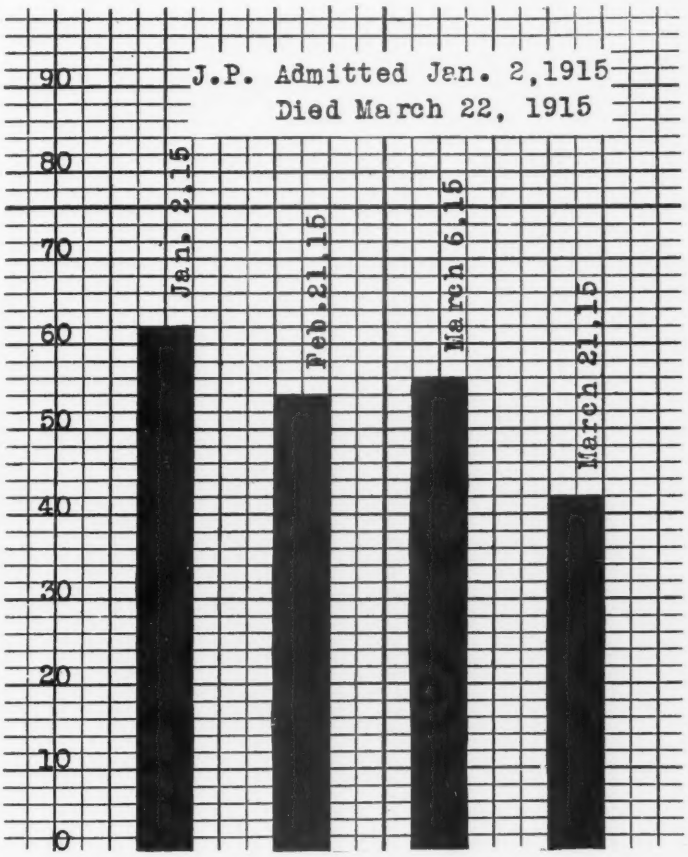

CHART X

bilateral pulmonary tuberculosis. Sputum positive for tubercle bacilli. Urine on admission showed trace of albumin with hyaline and granular casts. No oedema, no uraemic symptoms. Function reduced, but not seriously impaired, and continued so to end. Death due to progressive asthenia.

CONCLUSIONS

1. The renal function in incipient pulmonary tuberculosis is good.

2. The renal function in advanced cases of pulmonary tuberculosis is reduced only when there is evidence of structural damage to the kidney. The impairment of function is not such as to be of itself of serious prognostic import.

3. The presence of albumin and casts in the urine with the consequently graver prognosis suggests that the kidney is participating in a generalized destructive process involving the anatomical integrity of the tissues of the body and that in spite of this the functional efficiency is maintained to a fair degree to the last days of life.

4. Death due to renal insufficiency is rare in pulmonary tuberculosis in spite of the frequent structural changes in the kidney in the last six months of life.

5. Our studies with regard to the diet have shown no reason why all types of tuberculous patients should not be abundantly fed. However, when evidences of a renal lesion were present and the function at the minimum normal point, or slightly below, we have reduced the protein intake and increased correspondingly the carbyhydrates and fats. Certainly protein excesses (as one occasionally sees in the giving of many eggs) should be avoided.

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WHAT MASSACHUSETTS IS DOING TO PREVENT AND CONTROL TUBERCULOSIS

JOHN B. HAWES, II

There are various ways by which any large community may try to solve its tuberculosis problem, and of these Massachusetts seems to have tried all. The principal ones are as follows:

1. To treat and if possible cure the early case by means of sanatoria.

Massachusetts, as a state, starting its antituberculosis campaign in 1898, adopted this policy. According to this method the community devotes its efforts towards the arrest and cure of the incipient consumptive.

2. To segregate and isolate the advanced consumptive in hospitals and other suitable institutions.

According to this method, the patient in the incipient stage, who is rarely a danger to the community, is more or less left to his own devices, to be cared for in private sanatoria or at home. The advanced consumptive with positive sputum is the object of attack. This second method means *segregation* rather than cure.

3. To educate the general public, to train the doctors in early diagnosis, and to supervise the patients by means of dispensaries and outpatient departments. These dispensaries should play an important rôle in any antituberculosis campaign. They are likely to be the first steps taken by a community in antituberculosis work. Alone they can do much; but associated with hospitals and sanatoria they can do much more. Education of the public and of the medical profession in the early diagnosis of tuberculosis rather than cure or segregation is the most important feature of this method.

4. *The intelligent use of all the above mentioned methods.* This is really what Massachusetts is trying to do. Such a combination of sanatoria for early cases, hospitals for advanced cases, dispensaries to discover the incipient consumptive and to disseminate information means not only treatment, cure, and segregation of tuberculosis but its *prevention.*

In handling such a problem as this, it is important to know first of all the size of the task that confronts us. What we have to do in Massa-

chusetts is approximately as follows: In the year 1915 there were 5000 deaths from all forms of tuberculosis in this state. From this it may be computed that there are at least 10,000 persons in Massachusetts who are sick with tuberculosis in some form and who are in urgent need of treatment. This, then, is our problem: first, to care for the 10,000 who do need treatment, and second, to prevent numberless others, already infected but not at present in need of treatment, from becoming so.

What are we doing now for these 10,000 consumptives, and what are we going to do for them in the future?

At present, there are four state sanatoria situated at North Reading, Lakeville, Westfield and Rutland respectively. These four institutions provide about 1000 beds. In other state institutions, such as the State Infirmary at Tewksbury, the State Farm at Bridgewater, etc., there are about 500 beds. The various local tuberculosis hospitals provide approximately about 1200 beds, and private sanatoria and hospitals bring the total up to nearly 3000 beds. The average length of stay of any patient in our state sanatoria being about six months allows these 3000 beds to care for 6000 patients annually, leaving 4000 to be cared for in other ways, more and usually less satisfactory.

The evolution of the establishment of these various institutions, state and municipal, is an interesting one. In 1898 the Rutland State Sanatorium was opened. Massachusetts was the first state to build a state sanatorium, and in consequence the name of Rutland is known far and wide throughout the country. Only incipient cases, carefully selected by a few paid examiners, were admitted. In addition, each patient had to guarantee the payment of board at $4 weekly before admittance. Thus, only a comparatively small group of patients, selected for physical and financial fitness could be taken. Nevertheless, it was a step in advance.

From 1898 to 1907, Massachusetts as a state made comparatively little progress. The reason was evident. We were caring for a few incipient cases and perhaps curing a few individuals. We were making no attempt at *prevention*, and were doing very little in *segregation* or *education*. This became evident to a committee appointed by .the governor to study the subject, with the result that in 1907 a commission was named whose duty it was to build three new sanatoria for patients in all stages of the disease and to take general charge of tuberculosis work in the state. Dr. Arthur Tracy Cabot was chairman of

this board, a position he held until his death in 1914. Dr. Cabot gave a vast amount of time, energy, and far-sighted wisdom and skill to this work, unpaid and at the time unappreciated. The fact that Massachusetts as a state is well in the van in its campaign against tuberculosis is a fitting memorial to him.

These three new sanatoria, each containing 150 beds, were completed early in the year 1910. They were at once filled, and there soon developed a waiting list formed of patients who stood most in need of treatment, namely, advanced consumptives. According to the statute this commission, at the completion of these newer institutions, became a board of trustees and took over the management of the sanatorium at Rutland as well as the others. The method of admission to Rutland by special examiners was done away with, leaving this important duty to those who really should perform it, the medical profession of the state. Likewise, the unjust provision that board must be guaranteed by the patient was repealed. Those able to pay were made to do so, those unable to pay were not refused admittance on this account nor were they officially pauperized. The town or city from which the patient came was held responsible for the board if liable according to law. Otherwise the state paid the expense.

The opening of these new institutions was a great step in advance. Many of the patients admitted were in the curable stages though too sick for Rutland. Others who could not be cured were at least made comfortable and happy, while of still greater importance, a large number of advanced consumptives whose sputum harbored countless millions of tubercle bacilli were taken off the streets and away from their children, families and fellow workers.

We all heaved a sigh of relief when these three new sanatoria were completed. And yet our task was just begun. Although these new institutions were very accessible, and easily reached by one or two hours' travel from the large centers of population, it became increasingly evident that they would not and could not solve the problem of those most in need of treatment—the advanced and progressing consumptives. Although urged not to do so, patients of this type were constantly being taken home or were leaving of their own accord just when they were most likely to infect others and most in need of segregation. This situation led us to our next step.

The preliminaries to this were taken in 1910, and consisted of the passage of two important laws. The first was known as the "Tuber-

culosis Hospital Act," and the second the "Subsidy Act." The first of these demanded that every city in the state provide itself with a tuberculosis hospital or in other ways make adequate provision. To make this a less drastic measure, the second act was passed, providing for a state subsidy, of $5 per patient whose sputum contained tubercle bacilli, to those cities which already had or would build such hospitals officially approved by our board of trustees.

Although cities were slow to comply with this law, and although the state health department at that time was slow in enforcing compliance with it, city after city gradually fell into line and either built a hospital for itself or else made arrangements with some other city already provided with one. In 1910, Boston and Cambridge were the only cities that had tuberculosis hospitals of their own. At present there are approximately thirty out of the fifty-four cities in this state which are thus provided, though by no means adequately so, with hospital accommodations for their advanced and urgent cases of consumption.

This brings us up to the year 1915 and to the next stage in our evolution. The reasons for this were largely financial ones. Theoretically it had seemed like an admirable plan for each city to erect and maintain its own tuberculosis hospital or to combine with some other city in so doing. Practically, however, except in the larger centres of population, this plan was found to present certain difficulties. In the first place, it costs vastly more per capita to maintain a small hospital than a large one. It can readily be seen that the cost per patient in a small hospital of ten or twelve beds would be much higher than that in the Boston Consumptives' Hospital, for instance, which contains over 400 beds. Again, although the law specifically stated that towns and cities might combine in building and maintaining a hospital, this was found to be a difficult if not impossible thing to do. I know of very few instances where this arrangement has worked out satisfactorily to both parties. Furthermore, this law requiring a local hospital applied only to cities, and did not include towns and villages, nor help out in any way the scattered rural communities. The county hospital seemed the best solution of this problem.

This had already been tried and found to work successfully in one county in this state. Hampshire County contains only one city of large size, Northampton, but a large number of small towns and villages. The Hampshire County Sanatorium, centrally located at Leeds, seems to solve that particular problem very well indeed. This was

opened in 1914. It comes under the Subsidy Act mentioned above. The following year appropriation was made for the Barnstable County Sanatorium. This county includes the Cape Cod district and is made up entirely of small isolated towns and villages. Last year an elaborate act was passed, the County Tuberculosis Hospital Law, which directs the various county commissioners to erect hospitals for consumptives in those counties not already provided for. None of these has yet been constructed. The details of this act and the advantages and particularly the disadvantages of such a sweeping measure are beyond the scope of this survey. When completed, however, these new institutions will probably furnish a sufficient number of beds for our present needs. Thus we have our state sanatoria for early and curable cases, our local hospitals for advanced, progressive, and emergency cases, and our county sanatoria, present and future, for those patients who are unsuited for state or local institutions, and especially for those coming from communities not provided with other hospital accommodations. In addition to these, there are various private sanatoria and hospitals, furnishing additional beds, that need not be considered here.

With 10,000 consumptives in this state in need of institutional treatment, it would seem an easy task to fill these beds with the proper patients. Such, however, is not the case. Advanced consumptives there are in abundance: too many, in fact, so that at Rutland, which is intended for incipient cases only, there has never been more than 40 per cent of patients of this class, while its infirmary is crowded with those in the advanced and progressive stages. At North Reading, Lakeville and Westfield, where no hard and fast line is drawn as to incipiency, but only "curable" cases are desired, the proportion of incipient cases dwindles down to 5 or 10 per cent of the whole. In the meantime, with a waiting list of from two to three months for our state sanatoria, there are many vacant beds in various local hospitals.

It would appear, therefore, that the needs of the present situation demand two things: first, that the incipient consumptives be recognized and placed under proper treatment; and second, that the public and the medical profession be made to realize that our state sanatoria are not intended as hospitals and homes for advanced and dying consumptives but that these should be sent to nearby hospitals. While the accomplishment of these tasks is one of the functions of the various out-patient departments and dispensaries in the state, fundamentally it belongs to the medical profession. The "tuberculosis dispensary"

law, passed in 1910, which required that every city or town of 10,000 inhabitants or over must be provided with such a dispensary, was openly disregarded until 1915, when the present state health department enforced it. These dispensaries are already proving their value in discovering early cases. Their effect in arousing the public and the medical profession to demand that the advanced consumptive be cared for in an institution suited to his needs near his home and relatives and friends, will, I hope, be increasingly beneficial as time goes on. As yet, I admit that I can see little improvement in this direction, and as a result I am constantly confronted with the sorrowful task of telling patients, their friends, and often their physicians, that the case is too far advanced and cannot be considered a suitable one for a state sanatorium. In far too many instances, neither the patient nor his physician is willing to consider a local institution. In the discovery of early cases, however, we are certainly making progress although the superintendents of our sanatoria would hardly admit this. The various forms of welfare work, of occupational supervision and examination now being carried on by many large business concerns are of increasing assistance in discovering such early cases.

So far, I have spoken chiefly of the sanatorium patient. As is evident from the figures I presented, there is a large number of consumptives who do not and who cannot get sanatorium treatment, or, if they do so, it is for far too short a period. What becomes of this class? What is the state doing for those who never go to any institution, for those patients awaiting admission, and especially for those patients after they have been discharged from a sanatorium?

The first group, those who never go to any institution, is an unfortunate one. Aside from a very slight and inadequate supervision carried on by the state health department, and by local boards of health, as far as the State of Massachusetts is concerned I can safely say that nothing is being done. The second class, those who are intending to go to a sanatorium but who, owing to the length of the waiting list, have to wait two or three months or more before they can be admitted, is better off. The mere fact that an application has been filed at the State House, and that the patient's friends and physician are aware that his admission depends somewhat on physical condition when his turn is reached, serves as a stimulus, in many instances, to both patient and doctor. Our dispensaries, and especially the nurses connected with such dispensaries are in a position to be of immense assistance to this group of cases.

The third class is the most important one of all. This is composed of ex-sanatorium patients. The state has invested a very large sum of money in these men and women. Whether this money is well invested or wasted depends on how these patients behave, or rather how they are made and allowed to behave after their discharge. In Massachusetts we feel that the doctor's responsibility for his patient does not end when he has sent him off to some sanatorium; nor do we feel that the responsibility of the state ends when the patient is discharged from some institution; nor again do we feel that at any time during this process, before, during his stay at the sanatorium, or after his return does the responsibility of the local health board end in this matter. It ends only when the patient is *cured*, which rarely happens; or when he is dead and his family is proved to be healthy and properly cared for.

The care of the discharged sanatorium patient is one of the great problems that confronts us. We have almost enough sanatoria and hospitals, but we do not pay enough attention to the patients after they leave the sanatorium. In 1912 we started our after-care work with one worker, Miss Bernice Billings, who had had training in both nursing and social service. It was her duty to visit each patient as soon as possible after discharge from a sanatorium. It is easy to imagine what an overwhelming task this was at first. Gradually, however, as local tuberculosis associations and particularly local dispensaries and boards of health realized and accepted their responsibility in regard to these discharged patients, her work has been made a little easier and less discouraging. There is still a vast amount to be done in this direction, and there is no field of work which will better repay any efforts spent on it.

So much for what we have done and are doing now. What progress are we making and what are our plans for the future? As to actual figures, I can show but little tangible proof that Massachusetts is curing consumptives. Miss Billings, our after-care worker, has recently looked up the present condition of 1056 patients discharged from our state sanatorium during the period from May 1912 to May 1914. Of these patients on admission there were approximately 17 per cent incipient and 36 per cent moderately advanced and 47 per cent advanced. At the present time of these 1056 patients, 316 or 30 per cent are well and working. Of course this period is far too short a one on which to judge actual results. Nevertheless, it is, to me at least, a source of surprise that, out of a group containing so many ad-

vanced cases and so many of a very low grade of intelligence, even 30 per cent are able to work and earn their living. Although at times one cannot but feel discouraged and pessimistic, if we compare the situation now with what it was five years ago, there is no doubt but that we are making steady progress.

The death rate is constantly falling. In 1906, the mortality for tuberculosis in Massachusetts was 150 per 100,000. It is now approximately 130 per 100,000. In 1906 there were less than 1000 beds for consumptives: there are now over 3000 beds, and the number is increasing.

The medical profession is slowly but surely improving in its ability to recognize tuberculosis in its early stages. The general public is coming slowly to realize that not only must the incipient consumptive be recognized and treated, and the advanced consumptive segregated and cared for, but of still greater importance, that the careless and incorrigible consumptive must be looked upon as a grave menace to public health and treated as such.

My own ideas for the future, which I hope to live long enough to see realized in part, at least, would include the following:

Open-air schools for all children, instead of waiting as we now do until the child gets sick before we give him the fresh air he needs. The stupid policy to which we still adhere in this regard I trust in ten years will be looked upon as a relic of barbarism.

A proper system of medical inspection and supervision of employees. Although a few broad-minded and far-sighted men now realize that the human machine is like any other machine in that it runs better and accomplishes more if kept in good condition, and that to keep it in good condition it must be periodically overhauled and inspected, far too many can see in this work only a foolish fad.

A proper workman's compensation law, combined with some form of health insurance which will do justice to the physician, adequately protect the workingman, and allow the insurance company a legitimate profit.

Tenement house laws and housing regulations which amount to more than mere words on a printed page and which are backed up and enforced by courageous and honest officials and supported by public opinion.

Laws, so framed that they can be enforced, to control the careless, ignorant, and incorrigible consumptive. We have plenty of laws at present, but they cannot be enforced and so are useless. To make them effective, the public must be educated to realize the need and justice of such laws and to demand that they be enforced.

EXPERIMENTAL INVESTIGATIONS OF TUBERCULOUS PERITONITIS AND THE EFFECT OF PNEUMOPERITONEUM

R. G. PESCHMAN AND H. J. CORPER

From the Laboratory of the City of Chicago Municipal Tuberculosis Sanitarium.

Tuberculous peritonitis with its many phased pathological and clinical manifestations is a subject of an admittedly intricate and complex nature. A great deal has been written and much experimental work carried on in attempts to solve various phases of the problem regarding its etiology and therapy. The literature is exhaustive on information regarding various types and methods of treatment applying every possible physical and chemical means from simple laparotomy, on the one hand, as an example of the former, to camphor, on the other hand, as an example of the latter.

Up to the present time the physical procedure, abdominal section and surgical interference have proved beneficial and successful. An explanation for these results has been sought ever since the pioneer observations on the first case in 1862 reported by Spencer Wells. Shortly after this time, two schools were established whose beliefs differed as to the proper method of procedure in cases of tuberculous peritonitis, the one favoring surgery as the treatment *per se*, while the other preferred the conservative hygienic dietetic method using various extra-abdominal medications without surgical interference. On the side favoring surgical procedure were allied such men as Koenig (1), Lindner (2), Roersch (3) and Frank (4); and on the conservative medical side, Borchgrevink (5) and Rose (6). Expectant treatment was shown by these observers to be just as beneficial as surgical intervention in the cases reported up to 1900. From then on, however, the adherents of the surgical doctrines brought forth more proof of the efficacy of their treatment in the form of cases improved, and at the present time abdominal section holds first place as a method of choice.

How does simple laparotomy influence the course of tuberculous peritonitis? Is it the mere operation of opening the abdomen, is it the exposure of the peritoneum to the air or one of its constituents, is it the light, the hyperaemia caused by the irritation of the peritoneum, the

alteration of the circulation, the stimulation to the formation of antibodies, or the change in intra-abdominal pressure that is the deciding factor in producing this effect? Martens (7) after reviewing the literature comes to the conclusion that no one of these agents in itself produces an effect. But the fact remains that abdominal section does in a great number of cases cure or at least limit the progress of the disease. Haberer and Clairmont (8), in a report on the reaction of the peritoneum in healthy and diseased animals, found that the injection of sterile urine (normal and pathological) or of sterile gastric or intestinal contents did not influence the absorption of potassium iodide by the peritoneum, while increased peristalsis hastened this. Single injections of air also did not alter the absorption by the peritoneum. In early peritonitis the absorption was hastened, while later the rate was decreased. After dry laparotomy the rate of absorption diminished while moist laparotomy produced no effect or only a slight one. Ligating or packing off the omentum caused a marked diminution of the absorption. Peritoneal transudates were not influenced by laparotomy.

Shipley and Cunningham (9) in recent investigations found that true solutions, pseudosolutions and fine granules were removed from the peritoneal cavity by the omentum, this occurring by way of the blood vessels, since true lymphatics are not demonstrable in the omentum. Gatti (10), in his paper on the histological changes occurring in peritoneal tuberculosis after simple laparotomy, found tuberculous peritonitis of the fibroid type improved in guinea pigs but this was not true of the caseous form. In rabbits and dogs there was even a tendency to heal when caseation had taken place. Moorhof (11) in 1893 began to use air in tuberculous peritonitis contending that this procedure kept the serous surfaces apart thus preventing adhesions and also aiding the absorption of fluids. This was followed by favorable reports on the use of air by Nolen (12), Folet (13) and a large number of other observers including the recent report by Brückner (14). Many Italian observers, including Rouet (15) and Floris (16), recommend treating tuberculous ascites by aspiration and replacing the fluid by air, thus obtaining favorable results. Among Americans who used gases injected intraperitoneally and obtained favorable results may be mentioned McGlinn (17) who, however, used oyxgen in place of air.

On account of the extensive use of artificial pneumothorax in the present day treatment of pulmonary tuberculosis and the beneficial effects derived therefrom, it was thought advisable to obtain more information

regarding the effect of this procedure upon the normal serous surfaces and upon those in contact with tubercle bacilli with a view to explaining some of the changes occurring in cases of pulmonary tuberculosis having had an artificial pneumothorax established. Most conspicuous of these changes found at autopsy is the pronounced thickening of the pleura, both parietal and visceral, and, after prolonged administration, the consequent increased time required for the absorption of the gas introduced. Since the pleura, peritoneum and pericardium are embryonically derived from the same primary lateral mesoblastic tissue (18) and are anatomically and histologically identical, it was deemed advisable, on account of the greater ease of experimentation to study the effects of pneumoperitoneum in rats, such animals not requiring for this purpose elaborate apparatus and being easily handled in large numbers for making Roentgenographs.

EXPERIMENTAL PART

In carrying out the following experiments several important points had to be observed. In the first place it was desirable to know the length of time required for the complete absorption of the air after it had been injected and whether the rate of absorption after the first injection differed from that after subsequent injections. Observations on this point were easily made by taking a series of Roentgenographs at short intervals and noting when the gas had entirely disappeared; thus it was a simple matter to keep up the inflation and also to study the speed of absorption. In the second place, in order to have comparable results, and a work of this type must of necessity be comparative with regard to the dissemination and amount of tuberculosis produced, dead tubercle bacilli were used since they produce all the lesions obtainable with the live organisms and in equivalent amounts always produce approximately equivalent amounts of tuberculosis. The uncertain growth factors of the bacilli and the individual resistance of the experimental animal to their development are thus eliminated.

PROTOCOLS

Series I. This series consists of thirty full grown rats which were apportioned into three separate experiments. One set was injected intraperitoneally with air alone to determine the influence of the gas upon the normal peritoneum, a second set was injected intraperitoneally with a heavy emulsion

of dead tubercle bacilli to determine the location attained by the bacilli and their effect upon the peritoneum, and the third set was injected with dead tubercle bacilli in an identical manner as set 2 and treated by immediate intraperitoneal injection of air, the inflation being kept up by frequent injections as in set 1. One rat of each set was killed and studied at various intervals after the initiation of these experiments.

EXPERIMENT 1

Air Injections Alone

This series received air injections during the period of the experiment at three to four day intervals in amounts of 20 cc.

Rat 1. Intraperitoneal air inflation was kept up for two weeks after which, on post mortem examination, the animal revealed no adhesions in the abdomen, no macroscopic changes in the parietal or visceral peritoneum and the omentum was free and to all appearances normal.

Rat 2. Intraperitoneal inflation was kept up for three weeks after which time post mortem examination revealed slight adhesions between the omentum and peritoneum at the site of the injections. Otherwise the omentum and peritoneum appeared normal.

Rat 3. Intraperitoneal air inflation was maintained for four weeks, after which time no changes were found in the abdomen except a slight adhesion of the omentum at the site of injection of the air similar to that found in rat 2.

Rat 4. After five weeks' inflation with the air the peritoneum and omentum were found normal and without adhesions at the site of injection.

Rat 5 after six weeks' inflation and rat 6 after ten weeks' inflation revealed a normal peritoneum and omentum except at the site of injection where a definite but slight thickening and fibrosis were noted, probably as a result of the frequent punctures.

Rat 7 after twelve weeks', rat 8 after fourteen weeks', and rats 9 and 10 after sixteen weeks' inflation revealed no abnormalities of the peritoneum or omentum except a slight thickening at the site of the punctures. Measurements of the thickness of the peritoneum by means of a micrometer caliper at a distance from the sites of injection revealed no appreciable difference in the cases where inflation had been kept up for sixteen weeks as compared with those not inflated.

EXPERIMENT 2

Injections of dead tubercle bacilli alone

This series of rats received intraperitoneally a single injection of a heavy suspension of dead tubercle bacilli (1 cc. in every case), the needle being

inserted in the left lower quadrant of the abdomen, and they were examined at definite intervals.

Rat 1. Two weeks after injection of the dead tubercle bacilli post mortem examination revealed no abnormalities at the site of injection on the parietal surface of the peritoneum. The peritoneum as a whole was smooth and glistening and all the organs, except the spleen and omentum, appeared normal. The spleen was partly surrounded by the omentum, the latter also enveloping a large mass at the lower pole of the spleen which on section proved to be a well encapsulated collection of tubercle bacilli with central caseous material about 1.5 mm. in diameter.

Rat 2. This rat was autopsied three weeks after injection of tubercle bacilli and revealed no abnormalities in the abdomen except that the omentum was found in the region of the stomach and was nodular to the touch. These nodules upon section proved to be the tubercle bacilli in a caseous center, well surrounded by a connective tissue capsule. The spleen was not involved in this mass.

Rat 3 was sectioned after four weeks and revealed a slight fibrinous exudate in the pelvis. The omentum was nodular and located at the lower pole of the spleen encapsulating it almost completely. These nodules had fairly thick connective tissue capsules in which the bacilli were found in a caseous mass. Otherwise normal findings obtained in the abdomen.

Rat 4 was autopsied five weeks after injection and revealed involvement of the spleen and a tuberculous mass 2.5 mm. in diameter surrounded by the omentum and a thick connective tissue capsule. All the other organs in the abdomen were normal. The spleen was studded with small caseous tubercles.

Rat 5 after six weeks, rat 6 after ten weeks, rat 7 after twelve weeks and rat 8 after fourteen weeks revealed very little difference from the previous rats except in minor details. In some cases the involvement in the tuberculous mass would extend to the liver or occasionally an adhesion to the parietal peritoneum would be found, and in others the spleen was more involved.

Rat 9. (Sixteen weeks.) The omentum encapsulated, a large tuberculous mass extending to the lower pole of the spleen and involving this organ. There was a single adhesion between the omentum and the parietal peritoneum. Otherwise the findings were normal.

Rat 10. (Sixteen weeks.) The entire spleen was involved in omental adhesions extending to a large mass on the lower surface of the liver between the liver lobes. The tuberculous mass was in the process of softening and discharging caseous material.

EXPERIMENT 3

Intraperitoneal injection of dead tubercle bacilli as in experiment 2 and the simultaneous inflation with air which was repeated at frequent intervals throughout the experiment

This series of rats received a single intraperitoneal injection of 1 cc. of a heavy suspension of dead tubercle bacilli indentical to that used in experiment 2 and in the same manner, and were immediately given 20 cc. of air intraperitoneally as in experiment 1, the inflation being repeated as in experiment 1 every three to four days.

Rat 1. This animal was sectioned two weeks after the injection of tubercle bacilli and air, and revealed a large number of small tubercles on the surface of the liver, spleen, and the intestines, the latter being adherent to the omentum in a number of places. The spleen was slightly enlarged. Otherwise the findings were normal.

Rat 2. After three weeks' inflation this rat revealed numerous small superficial tubercles on the spleen, and the omentum surrounded numerous tuberculous masses in the region of the spleen and greater curvature of the stomach. The peritoneum was not markedly changed in appearance except at the site of the injections of air, which location was slightly hyperaemic.

Rat 3, autopsied four weeks after the initiation of the experiment, revealed numerous small tubercles on the under surface of the liver and some gray tubercles on the surface of the spleen. The omentum surrounded numerous tubercles some as large as 1 cm. located below the stomach and adjacent to the spleen. The diaphragm and peritoneum were normal.

Rat 4. This rat died about the fifth week and revealed the peritoneal cavity filled with a sanguineous fluid. The omentum was folded on the under surface of the liver and surrounded numerous large tubercles. The lower surface of the liver was studded with miliary tubercles and the spleen was soft and contained many superficial small tuberculous nodules. The peritoneum at the site of injection was dull.

Rat 5 after six weeks and rat 6 after ten weeks revealed tubercles on the surface of the liver and spleen with marked involvement of the latter by the tuberculosis. Tubercles were also found on the abdominal surface of the diaphragm and a few were scattered over the peritoneum.

Rat 7, autopsied after twelve weeks was apparently normal, having received an injection of tubercle bacilli into the pleural cavity through the diaphragm.

Rat 8 after fourteen weeks revealed many adhesions between the omentum and the liver, some of the large tubercles in the omentum beginning to soften. Numerous tubercles were found on the abdominal surface of the diaphragm

and the peritoneum in general. The peritoneum was dull and congested at the site of injection. The spleen was full of superficial tubercles.

Rat 9 after sixteen weeks revealed small tubercles on the surface of the liver and spleen and some nodules in the mesentery. The omentum was adherent to the peritoneum in a number of places and contained numerous tubercles.

Rat 10 after sixteen weeks revealed the omentum adherent to and encapsulating a large tuberculous mass below the stomach and adjacent to the spleen. The spleen was buried in adhesions and the liver contained numerous superficial tubercles. The intestines contained numerous small tubercles and there was a fibrinous exudate on the peritoneum. The abdominal surface of the diaphragm contained some well defined tubercles.

SUMMARY OF SERIES I

As a result of the above experiments the following conclusions seem justified:

1. The intraperitoneal injection of air into normal rats produces no visible alterations in the peritoneum or abdominal organs even if the inflation is repeated at three or four day intervals for a period of sixteen weeks.

2. Dead tubercle bacilli injected intraperitoneally into normal rats (needle inserted into lower left quadrant of the abdomen) are usually found well walled off by the omentum in the vicinity of the greater curvature of the stomach and the spleen, which organ is generally involved in this walling off.

3. Dead tubercle bacilli injected intraperitoneally into rats coincident with the inflation with air, which inflation is repeated at three or four day intervals for periods up to sixteen weeks, reveal a marked dissemination of the tubercle bacilli in the peritoneum. This dissemination is especially pronounced on the under surface of the liver, the abdominal surface of the diaphragm and occasionally the intestines in general. The air itself had no appreciable effect upon the peritoneum in such animals.

Series II. Effect of intraperitoneal air inflation upon the rate of absorption of the air in normal rats and in rats with tuberculous peritonitis

In series I no special attention was paid to the effect of the intraperitoneal air inflation upon the subsequent absorption of air in normal rats or of tuberculous peritonitis upon such absorption.

With a view to determining these facts a series of six rats was used, one set, as in series I, receiving air alone, another set dead tubercle bacilli alone and a third both tubercle bacilli and air. At frequent intervals the rats were X-rayed daily to determine the presence of air in the peritoneal cavity, thus to note the rate of disappearance of the air and to inject air so that an inflation was kept up throughout the course of the experiment.

EXPERIMENT 1

The injection of air intraperitoneally

Rats 1 and 2 were normal rats. They were X-rayed before the experiment (see fig. 1) as controls and then 20 cc. of air was injected intraperitoneally and the rats were again X-rayed. The appearances noted are shown in fig. 2. The rats were then X-rayed daily thereafter and the findings noted as seen in figs. 3, 4, and 5. It is observed that in fig. 5 at the end of three days there is an absence of air in the peritoneal cavity, thus indicating that the 20 cc. of air disappeared upon primary injection into a normal rat within three days. At the end of four months, during which time the inflation was kept up at three days intervals controlled by X-ray examination the rate of absorption of the air was found not to have appreciably changed. Post mortem examination at this time revealed no abnormalities and the findings were similar to those in series I.

EXPERIMENT 2

The injection of tubercle bacilli and the subsequent injection of air at the end of four months

In this experiment were included two rats, one receiving 1 cc. of a heavy emulsion of dead tubercle bacilli intraperitoneally and the other 2 cc. of the same emulsion. At the end of four months 20 cc. of air was injected intraperitoneally and the rate of absorption watched with the X-ray as in experiment 1. It was noted that absorption occurred, as in experiment 1, in three days indicating that the tubercle bacilli injected intraperitoneally had no influence after four months upon the absorption of the air. The post mortem findings in these rats were identical with those in series I, experiment 2.

FIG. 1. X-RAY PHOTOGRAPH OF NORMAL RAT BEFORE THE INJECTION OF AIR

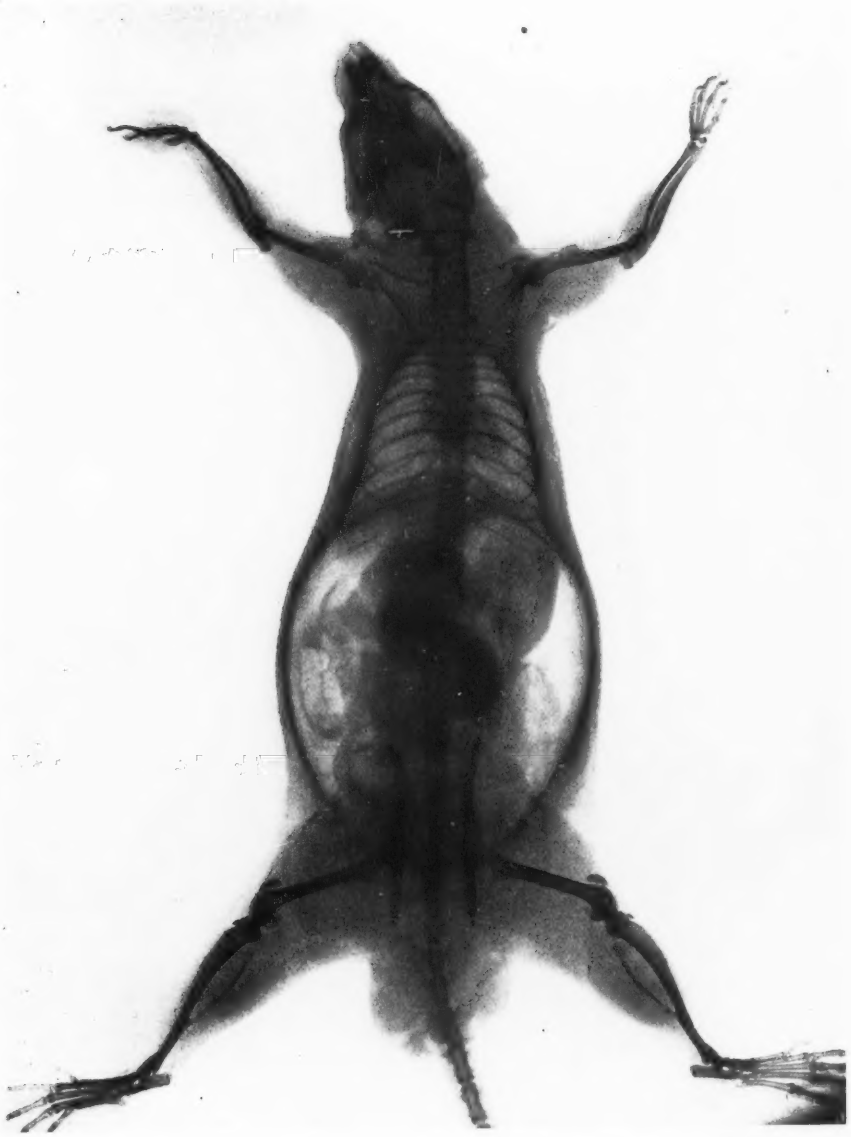

FIG. 2. X-RAY PHOTOGRAPH IMMEDIATELY AFTER THE INTRAPERITONEAL INJECTION OF AIR

FIG. 3. X-RAY PHOTOGRAPH ONE DAY AFTER THE INTRAPERITONEAL INJECTION OF AIR

FIG. 4. X-RAY PHOTOGRAPH TWO DAYS AFTER THE INTRAPERITONEAL INJECTION OF AIR

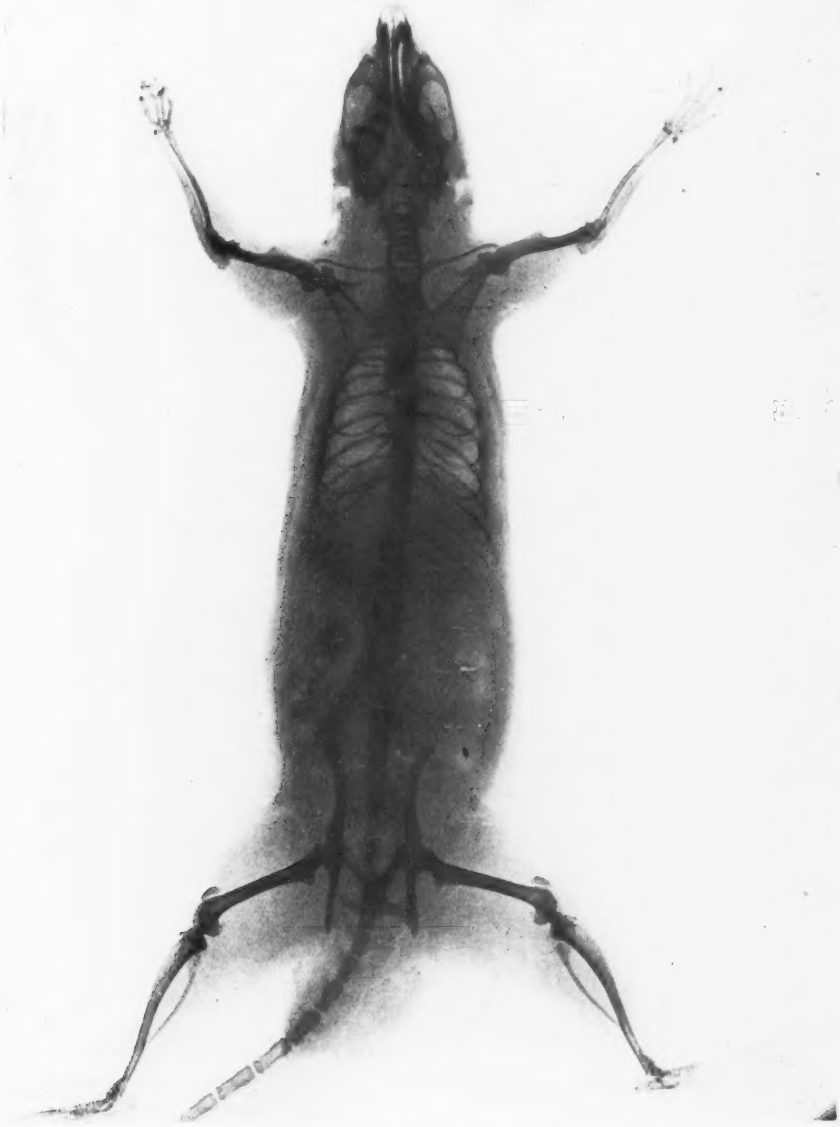

FIG. 5. X-RAY PHOTOGRAPH THREE DAYS AFTER THE INTRAPERITONEAL INJECTION OF AIR

EXPERIMENT 3

The injection of tubercle bacilli intraperitoneally, and immediate intraperitoneal inflation with air which was kept up by repeated injections

Two rats were used. One received 1 cc. of a heavy emulsion of dead tubercle bacilli and the other 2 cc. of the same emulsion, and both were given an immediate injection of 20 cc. of air intraperitoneally, the inflation being maintained by frequent injections of air every three days, as indicated by the X-ray. The rate of absorption of the air from the peritoneal cavity revealed no difference at any time during the course of four months of inflation. At post mortem examination the findings were almost indentical to those in series I, experiment 3, except that the amount of tuberculosis was slightly more widespread and greater in amount.

SUMMARY OF SERIES II

Rats that received an intraperitoneal injection of dead tubercle bacilli revealed no difference in the rate of absorption of air, injected intraperitoneally only once four months after the bacilli had been injected, as compared to those in which the absorption took place initially or at any time during the four months inflation by repeated intraperitoneal injection of air.

Series III. Clinical observations on the absorption from the pleural cavity in cases of pulmonary tuberculosis with pneumothorax.[1]

In an attempt to study the rate of absorption from the pleural cavity in cases of pulmonary tuberculosis treated with artificial pneumothorax of different durations, a series of five cases were carefully observed with regard to the rate of absorption of the air and also the rate of absorption of methylene blue from the pleural cavity.

Case I, Patient H., entered the hospital July 20, 1916, and a diagnosis of spontaneous pneumothorax with pleural effusion was made. The fluid increased in amount with no discomfort to the patient and was not removed. X-ray examination revealed an absolute hydrothorax free from gas during this time. On December 3, 1916, 1100 cc. of fluid were removed and 850 cc. of air injected. Twenty-four hours later an additional 300 cc. and seventy-two hours later another 100 cc. of air were injected. After the last injection of air 10 cc. of a 5 per cent solution of sterile medicinal methylene blue were

[1] We wish to acknowledge our appreciation to Dr. Imas P. Rice for carrying out the experiments and supplying the following cited data.

injected into the pleural cavity and eight hours later the first traces of methylene blue were found in the urine, the excretion continuing twenty-eight days.

Case II, Patient A., entered the hospital July 7, 1916, with tuberculous infiltration of both apices and became progressively worse until cavity formation was found at the right apex, verified by X-ray examination. On November 10, 1916, the right lung was completely collapsed (no adhesion being present) by means of air. At the beginning the air was injected every two or three days. The time was later lengthened as necessity required to every seven days. The average amounts necessary to give positive pressure were about 550 cc. On December 11, 10 cc. of 5 per cent medicinal methylene blue were injected into the pleural cavity and five hours later it appeared in the urine and was excreted for about six days.

Case III, Patient K., entered the hospital on June 8, 1916, having had an artificial pneumothorax performed two months previous to admission, Physical and X-ray examinations revealed a partial pneumothorax with adherent uncollapsed cavity at the right apex. Refillings of air were made every seven days, the first three months after admission required about 300 cc. and the subsequent three months about 200 cc. to maintain a satisfactory pneumothorax. On December 11, the patient received 10 cc. of 5 per cent medicinal methylene blue into the pleura which appeared in the urine eight hours after the injection and was excreted for about ten days.

Case IV, Patient F., entered the hospital June 23, 1915, and upon examination revealed râles at both apices, which condition, especially on the right side, became progressively worse. The patient later began to bleed from the right lung and for this reason was completely collapsed on December 5, 1915, with about 1800 cc. of air. Refillings were then made every three days for about two weeks, after which they were made every seven days, averaging about 500 cc. for the first half year and 400 cc. for the second half year. On December 8, 1916, 10 cc. of 5 per cent medicinal methylene blue was injected into the pleural cavity and it appeared in the urine three hours after injection and persisted for about nine days.

Case V, Patient G., entered the hospital March 12, 1915, having had pneumothorax performed four months previously for haemorrhage, and upon examination revealed partial pneumothorax on the left side with uncollapsed multilocular thick walled cavity in the upper uncollapsed portion. The refillings of 250 cc. were given every seven days, and on December 8, 1916, an injection of 10 cc. of 5 per cent medicinal methylene blue was given into the pleura. This appeared in the urine two hours after injection and persisted for three weeks.

DISCUSSION

From the observations made by previous investigators, including Klemperer (19), and those cited in the above cases, it can be considered an established fact that the absorption of the gas diminishes as the time of persistence of the pneumothorax increases. Rest (20) in a good resumé of pneumothorax also considers this an established fact. With regard to the absorption of crystalloids (methylene blue) from the pleural cavity it can be stated that, as the duration of the time of inflation increased, at least no increase in the time necessary for the absorption into the general circulation, as indicated by its appearance in the urine, was noted. Now wherein lies the discrepancy between the absorption of the gas from the pleural cavity, where a diminution in rate occurs as the time of persistence of the pneumothorax increases, as compared to that occurring in the peritoneal cavity, where no change in rate of absorption is noted as the time of persistence of the pneumoperitoneum increases. Since the pleura and peritoneum do not differ greatly in fundamental properties, some other explanation must be sought for than the mere effect of the gas *per se*.

That there is a fibrosis of the lung resulting from artificial pneumothorax was noted by Warnecke (21) who called it cirrhosis of the lung. It was produced experimentally in normal dogs by Kaufmann (22). Kistler (23) pointed out that there is an unmistakable and direct connection between this fibrosis and the pressure exerted by the pneumothorax. But whether this fibrosis *per se* affects the rate of the absorption of the gas in the pleural cavity is still to be answered. It does not diminish the absorption rate of crystalloids. As a result of the above observations we are inclined to believe that the decreased rate of absorption can probably be accounted for by the fact that we are dealing rather with a chamber, the rigid chest wall, in which is enclosed the lung which, when pneumothorax is first performed, tends to reëxpand and by this tendency aids the rapid removal of the gas from the pleural cavity. Later on, however, as the pneumothorax is kept up, on account of factors such as the fibrosis, etc., the lung tends to remain collapsed and this would in itself diminish the rate of disappearance of the gas from the pleural cavity. On the other hand in pneumoperitoneum the conditions are entirely different. The pressure, which in pneumothorax causes the fibrosis probably indirectly, is compensated for by the flexible abdominal walls and thus the possibility of any change in absorp-